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**UNITED STATES  
SECURITIES AND EXCHANGE COMMISSION**

Washington, D.C. 20549

**FORM 10-K**

(Mark One)

☒ **ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE  
SECURITIES EXCHANGE ACT OF 1934**

**For the fiscal year ended December 31, 2001**

**OR**

☐ **TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF  
THE SECURITIES EXCHANGE ACT OF 1934**

**For the transition period from \_\_\_\_\_ to \_\_\_\_\_**

**Commission file number 033-76414**

**ARIAD Pharmaceuticals, Inc.**

(Exact name of registrant as specified in its charter)

**Delaware**

**22-3106987**

(State or other jurisdiction of  
incorporation or organization)

(I.R.S. Employer Identification No.)

**26 Landsdowne Street, Cambridge, Massachusetts 02139-4234**

(Address of principal executive offices)

(Zip Code)

**Registrant's telephone number, including area code: (617) 494-0400**

**Securities registered pursuant to Section 12(b) of the Act: None**

**Securities registered pursuant to Section 12(g) of the Act:**

**Common Stock, \$.001 Par Value**

**Rights to Purchase Series A Preferred Stock**

Indicate by check mark whether the registrant (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days.

YES ☒ No ☐

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K is not contained herein, and will not be contained, to the best of registrant's knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. ☒

The number of shares of the registrant's Common Stock outstanding as of March 18, 2002: 32,418,371.

The aggregate market value of the voting stock held by nonaffiliates of the registrant was approximately \$143 million as of March 18, 2002, based on the last reported sales price of the registrant's Common Stock on the Nasdaq National Market on such date.

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## PART I

### ITEM 1: BUSINESS

The following Business section contains forward-looking statements, which involve risks and uncertainties. Our actual results could differ materially from those anticipated in these forward-looking statements as a result of certain factors (see “Certain Factors That May Affect Future Results of Operations”). In this Annual Report on Form 10-K, we incorporate by reference certain information from parts of other documents filed with the Securities and Exchange Commission, or the SEC. The SEC allows us to disclose important information by referring to it in that manner. Please refer to such information when reading this Annual Report.

#### Corporate Overview

We are engaged in the discovery and development of breakthrough medicines that regulate cell signaling with small molecules. Breakthrough medicines are products, created *de novo*, that may be used to treat diseases in innovative ways. We currently have five lead product candidates in our development pipeline, including: (1) our drug candidate, AP23573, that controls cell proliferation and nutrient uptake to tumors to treat cancer; (2) our bone-targeted drug candidate, AP23451, to treat the complications of cancer that has spread to bone, or bone metastases; (3) our regulated protein therapy product candidate to treat anemia in which the production of erythropoietin is precisely controlled *in vivo* using an orally administered drug, AP22594; (4) our T cell immunotherapy product candidate in which a non-immunosuppressive drug, AP1903, may be used to treat graft-vs-host disease, or GvHD, following donor bone marrow transplantation – a therapy for cancer and other immune and blood diseases; and (5) our dual-action drug candidates, AP23485 and its analogs, that block bone resorption and stimulate bone formation to treat osteoporosis. Our product candidate for GvHD is in phase 2 development. Our product candidates for cancer, bone metastases and anemia are at the pre-investigational new drug, or pre-IND, stage of development, and our drug candidates for osteoporosis are in preclinical development.

With respect to the development and commercialization of our current lead product candidates, we intend to: (1) develop our current lead product candidates at least through phase 2 clinical trials; (2) establish the commercial infrastructure to market our portfolio of hematology and oncology product candidates in the United States; and (3) pursue a worldwide partner for our osteoporosis product candidate and partners for our hematology and oncology products outside the United States, generally after we are able to obtain phase 2 clinical data.

The NF- $\kappa$ B pathway has emerged as an important drug target in pharmaceutical research. This cellular protein and its associated cell-signaling pathways regulate genes involved in many major diseases, including atherosclerosis, asthma, arthritis, cancer, diabetes, infectious disease, inflammatory bowel disease, rheumatoid arthritis, sepsis, and stroke. We have an exclusive license to pioneering technology related to the discovery, development, and use of drugs that modulate the NF- $\kappa$ B pathway.

Our RegTech cell-signaling regulation technologies already are being used by over 500 academic investigators worldwide for scientific research and are the subject of over 120 published papers in the scientific literature. In return for providing these technologies for academic research, we receive certain intellectual property and commercialization rights to discoveries made resulting from their use. In effect, these researchers provide a robust source of potential new technologies, drug targets and product candidates that we may develop.

Our business plan aims to balance potential near-term revenues from licensing with longer-term product development. We are marketing licenses to our RegTech technologies and our NF- $\kappa$ B intellectual property portfolio to pharmaceutical and biotechnology companies to accelerate their genomics, proteomics and drug

discovery efforts. Commercial licenses have not been entered into as of yet. In addition, we may partner our cell-signaling regulation technologies for joint development of novel products, especially with companies that have proprietary therapeutic genes, cellular systems (e.g., stem cells) or gene delivery vectors.

ARIAD was organized as a Delaware corporation in April 1991. Our principal executive offices are located at 26 Landsdowne Street, Cambridge, Massachusetts 02139-4234, and our telephone number is (617) 494-0400. We maintain a web site at [www.ariad.com](http://www.ariad.com).

## **Our Lead Product Candidates**

We currently have five lead product candidates in our development pipeline, all of which are small-molecule drugs that regulate cell signaling. Many of the critical functions of cells, such as cell growth, differentiation, gene transcription, metabolism, motility and survival, take place through the processes of cell signaling. Disruption or over-stimulation of cell-signaling pathways has been implicated in many disease states. Our cell-signaling inhibitor programs are focused on discovering and optimizing small-molecule drugs that interfere with specific cellular proteins or pathways that have been well-characterized and validated as targets.

All of our lead product candidates were developed in-house through the integration of our major areas of scientific expertise: structure-based drug design and chemo-informatics, or computational chemistry, functional genomics and proteomics, and protein engineering. We believe that our lead product candidates will serve large, unmet medical needs.

The first four lead product candidates in our pipeline constitute our growing portfolio of hematology and oncology product candidates that we intend to commercialize in the United States. For those product candidates for which we are able to obtain phase 2 clinical data demonstrating initial evidence of efficacy and safety, we plan to pursue partnerships with major pharmaceutical or biotechnology companies (e.g., a worldwide partner for our product candidate for osteoporosis and partners for our hematology and oncology product candidates outside the United States market). Our lead product candidates are as follows:

<b><u>Product Candidate</u></b>	<b><u>Target</u></b>	<b><u>Initial Clinical Indications</u></b>	<b><u>Status</u></b>
AP23573	mTOR	Cancer (PTEN-deficient tumors)	Pre-IND
AP23451	Src	Bone metastases, Bone pain, Hypercalcemia	Pre-IND
AP22594 - ARGENT	EPO	Anemia (Multiple myeloma, Chronic renal failure)	Pre-IND
AP1903 - ARGENT	Fas	Graft-vs-host disease (T cell immunotherapy)	Phase 2 Development
AP23485	Src	Osteoporosis, Paget's disease	Preclinical Development

Preclinical development means that *in vivo* and *in vitro* studies are underway to designate a compound for clinical development. Examples of these studies include comparative safety and efficacy studies of lead compound analogs in disease models and formulation optimization. After clinical candidate designation, pre-IND development is initiated and consists of studies required by the U.S. Food and Drug

Administration, or FDA, or other regulatory authorities for inclusion in an IND or other regulatory filings to initiate clinical studies. Examples of these studies include toxicology, pharmacology, and metabolism studies conducted under the current Good Laboratory Practices, or cGLP, requirements. Clinical development requires manufacturing of clinical-grade material (e.g., small-molecule drugs and gene-transfer vectors) produced under the current Good Manufacturing Practices, or cGMP, requirements. Phase 2 development includes obtaining regulatory and institutional review board, or IRB, approvals for administering product candidates to patients with disease and conducting clinical trials that are designed to provide safety data and initial indications of a product's clinical efficacy in its proposed use. With respect to our GvHD product candidate, this may include patients with various types of blood cancers, various degrees of human leukocyte antigen, or HLA, mismatch, and various types of malignant and non-malignant diseases.

## **Cancer**

***The Disease:*** Cancer, the second leading cause of death in the Western world, is a complex collection of hundreds of separate diseases characterized by uncontrolled cell growth. Great strides have been made in the past few decades in understanding the molecular basis for this transformation by searching for genetic differences between normal cells and cancer cells. These studies have revealed that cancer cells often harbor genetic mutations that alter the controlling mechanisms in cell signaling that constrain cell growth in healthy cells. Two major classes of such genetic alteration have been identified. Genes that turn on cell growth and division are often over-activated or over-expressed in tumors. These genes are called oncogenes, and they represent key targets for anti-cancer drug design, since drugs that inhibit their activity should re-establish normal growth control. A second class of genes, called tumor suppressors, plays an opposite role by preventing unwanted cell growth. These genes are often inactivated or deleted in tumors, again leading to uncontrolled proliferation and potentially enhancing the effects of oncogenes.

***Current Therapies:*** Several forms of medical therapy have evolved as adjuncts or alternatives to surgery, including the introduction of cytotoxic chemotherapy and radiation therapy over 50 years ago. Although they continue to be the mainstay of cancer treatment, especially after the cancer has spread from its site of origin, these therapies are limited by lack of specificity and toxicity. Healthy cells, along with malignant cells, are killed by these agents. Recently, a number of alternative therapies have been introduced, including endocrine therapy to treat cancers of certain hormone-sensitive organs, recombinant biologics, and small molecules and monoclonal antibodies that target the molecular determinants of the transformation of normal cells to cancer cells. Several growth factor inhibitors and protein kinase inhibitors have been developed to block molecular pathways implicated in cancer.

***Our Approach:*** Using our expertise in structure-based design of drugs that regulate cell-signaling pathways, our scientists are engaged in several anti-cancer drug development programs. The most advanced program has led to the discovery of one of our lead product candidates, AP23573 – a potent small-molecule inhibitor of the protein kinase, mTOR, that plays a key role in orchestrating the progression of the cell cycle and controlling nutrient uptake to tumors. Genetic and other studies have demonstrated that blocking mTOR arrests tumor growth. This effect is even more pronounced in tumor cells with a deleted or mutated form of a tumor suppressor gene known as PTEN. Tumors with abnormalities in PTEN represent several of the major difficult-to-treat cancers, including prostate, uterine, pancreatic, and ovarian cancer, as well as melanoma, leukemia and gliomas.

Since cancer patients with PTEN-deficient tumor cells can be identified using genetic tests, it should be possible to select those who may benefit most from AP23573, which is currently in pre-IND development.

## Bone Metastases

**The Disease:** The spread of malignant cells from the primary tumor to the skeleton and the subsequent destruction of bone is a frequent and debilitating complication of many cancers, especially breast, prostate, and lung cancer and multiple myeloma. Bone metastases are a common cause of cancer-associated morbidity.

Like osteoporosis, bone metastases result in severe bone loss but also are associated with local pain, fractures, vertebral instability and compression, and elevations in blood calcium often to life-threatening levels. These clinical findings can be devastating to a cancer patient. Bone metastases are a major problem in cancer management, both in terms of cost and human suffering.

**Current Therapies:** There is no known cure for bone metastases. However, several therapies are used to treat bone metastases and skeletal-related events, including chemotherapy, hormonal therapy, radiotherapy, analgesics for pain and anti-resorptive drugs. Most recently, newer-generation bisphosphonates have been advanced for the treatment of bone metastases and have already achieved substantial worldwide sales for this indication. However, the efficacy of this class of agents in treating certain of these complications of cancer appears to decline during treatment.

**Our Approach:** Recent research demonstrates that most skeletal destruction due to bone metastases is mediated by osteoclasts – bone cells responsible for bone resorption. In fact, there appears to be a close relationship between metastatic cancer cells and osteoclasts. Cancer cells stimulate osteoclasts to increase their bone-resorbing activity, which, in turn, release bone-derived growth factors that both attract cancer cells to the bone surface and facilitate their growth and proliferation.

Advances in our understanding of the co-dependent relationship between osteoclasts and cancer cells have led us to a new preventive and therapeutic strategy – designing a potent inhibitor of bone resorption by down-regulating the cell signaling pathway that controls the activity of osteoclasts. We believe that inhibiting bone resorption through this mechanism will not only prevent the skeletal-related clinical events directly attributable to bone loss but also result in a less favorable local environment for tumor growth.

AP23451 is a small-molecule drug that potently inhibits a well-validated protein tyrosine kinase believed to be implicated in the activation of osteoclasts. AP23451 is highly specific in targeting this kinase in bone. Its anti-resorptive activity has been repeatedly demonstrated in *in vivo* studies conducted by our researchers and corroborated by academic scientific teams. AP23451 is in pre-IND development.

## Anemia

**The Disease:** Red blood cells, produced in the bone marrow, transport oxygen from the lungs to the cells of the body and carbon dioxide to the lungs. Erythropoietin, or Epo, is a naturally occurring protein made primarily in the kidney that stimulates the manufacture of more red blood cells, when needed, by a process known as erythropoiesis. The role of Epo, therefore, is to maintain the number of red blood cells at an optimal level to provide sufficient oxygen transport to cells and tissues. If the amount of oxygen available to the cells is too low, a feedback mechanism stimulates the production of Epo and the manufacture of red blood cells. In addition, Epo stimulates the growth of stem cells in the bone marrow to become mature red blood cells. If the body loses its ability to manufacture sufficient quantities of Epo, the optimal number of red blood cells in the circulation no longer can be maintained. This is the case for many individuals who suffer from severe renal disease and results in a steady decrease in the number of red blood cells, which eventually leads to a decrease in the required transport of oxygen to tissues. A clinically significant reduction in the number of red blood cells (and their oxygen carrying component, hemoglobin) is known as anemia.

**Current Therapies:** Recombinant Epo (including a newer-generation version) is presently being used for the treatment of anemia caused by chronic renal failure (including end-stage renal disease), cancer chemotherapy and zidovudine treatment of HIV-infected individuals. Epo also has a role in the treatment of anemic patients undergoing elective noncardiac or orthopedic surgery, reducing the need for these patients to undergo pre-operative blood collections and post-surgical blood infusion. Today's standard treatment generally requires recombinant Epo to be injected on a recurring schedule into a vein or under the skin.

**Our Approach:** We are developing an alternative approach to deliver and regulate therapeutic proteins such as Epo based on our ARGENT cell-signaling regulation technology. We have selected Epo as our initial product candidate to demonstrate the clinical utility of this platform for orally regulated protein therapy. Rather than relying on repetitive injections of Epo to provide the therapeutic benefit, our approach provides a form of protein therapy regulated by small-molecule drugs. Our orally regulated protein therapy candidate is designed to involve a single, or infrequent, injection(s) of the Epo gene using a gene-transfer vector into the patient's muscle or other tissue in an inactive form. The patient then will take our orally administered drug, AP22594, which we believe will activate the Epo gene to manufacture the patient's own Epo. The production of Epo would only occur in response to the patient taking the drug, and the amount of Epo manufactured *in vivo* would depend on the amount of AP22594 the patient takes. The Epo produced by the genes in the body appears to work in the same way and have the same beneficial effects as Epo naturally produced by healthy kidneys. The ARGENT cell-signaling regulation technology may offer a means of precisely controlling the amounts of Epo to be delivered by adjusting the dose of the drug.

Worldwide sales for recombinant human Epo reached approximately \$6 billion in 2001, and substantial growth in the market is anticipated over the next five years as new products and more convenient dosing regimens are developed. This provides opportunities for new technologies that have potential benefits over the current products. We believe that the potential competitive advantages of our product candidate for anemia include:

- Replacement of an injectable recombinant product largely by an orally active drug administered infrequently;
- Protein production precisely controlled within a therapeutic window as opposed to the oscillating blood levels that frequently occur by injectable routes of administration;
- Sustained high levels of protein achievable, if desired, greater than those routinely obtained with the recombinant protein;
- Manufacturing costs minimized.

Our scientists have demonstrated regulated production of Epo in animals, including mice and non-human primates, for a duration of over three and one-half years. In addition to potentially providing improved medical care for patients with anemia, we believe this program will serve as an excellent model to demonstrate the clinical utility of our ARGENT system for the delivery of many different therapeutic proteins. Our AP22594 product candidate for anemia is currently in pre-IND development.

### **Graft-vs-Host Disease**

**The Disease:** Bone marrow transplantation, or BMT, followed by T cell immunotherapy, has become a well-established medical procedure to treat diseases that until recently were considered incurable. Such procedures are an important therapy today for numerous cancers, in particular hematologic malignancies such as leukemias. In addition, patients with solid tumors, such as breast and colon cancer, and non-malignant diseases, such as hemoglobinopathies and autoimmune diseases, have been shown to benefit from bone marrow transplants. In principle, the procedure permits patients to receive high doses of cytotoxic

chemotherapy and/or radiation therapy to kill abnormal cells within the bone marrow itself or abnormal cells at a site other than in the bone marrow. In either case, the aggressive treatment not only eliminates the unwanted (cancerous) cells but also destroys healthy cells within the bone marrow. Therefore, the patient's bone marrow must be replaced by infusion of bone marrow or peripheral stem cells from a donor, generally followed by T cell immunotherapy to help reconstitute the recipient's immune system.

There are approximately 40,000 bone marrow transplants performed annually. Over 35% of these are allogeneic (i.e., the bone marrow and T cells are obtained from a donor rather than from the patient's body). In approximately half of the recipients of donor bone marrow and T cells, the T cells attack the patients' own tissues, causing a disease known as graft-vs-host disease, or GvHD. If there is not a good genetic match between the donor and patient, the recipient has an even higher risk (as high as 90% depending on the degree of mismatch) of developing GvHD. This life-threatening complication is mediated by the ability of T cells to recognize an inherited set of genetic markers known as HLA that are found on the surface of human cells. If the donor's T cells recognize that the patient's HLA are sufficiently different from the donor's HLA, the donor's T cells quickly activate the immune system to destroy the recipient's cells, which are recognized as foreign. The major organs attacked in this process are the patient's skin, mucosa, liver and gastrointestinal tract. BMT patients also have an increased susceptibility to viral and bacterial infection until their immune system has reconstituted.

**Current Therapies:** The incidence and severity of GvHD can be reduced by withholding or limiting T cell immunotherapy following BMT. Unfortunately, this also eliminates the beneficial effects of those T cells, which include: (1) anti-tumor activity; (2) improved engraftment of donor bone marrow in the patient and immune reconstitution; and (3) prevention of early infectious complications by providing a functional cellular immune system. To recapture the beneficial effects of T cells, T cell depleted bone marrow often is supplemented with delayed infusion of donor T cells, referred to as a donor lymphocyte infusion or T cell immunotherapy. However, for these patients, GvHD represents a common and potentially lethal complication of the procedure.

Highly effective treatments for GvHD are currently unavailable. In fact, clinical experience indicates that approximately 50% of GvHD patients fail to respond fully to the current standard treatment which generally consists of immunosuppressive agents. Such drugs also put the patient at greater risk of infection, since they compromise an already weakened immune system. Although there are several alternate therapies under clinical investigation, including T cell directed monoclonal antibodies, and cytokine antagonists, we believe that they are limited by their inability to distinguish the T cells that are causing GvHD from those T cells providing beneficial effects. Consequently, these treatments may eliminate the beneficial effects of the immune cells that are being produced by the transplanted bone marrow.

**Our Approach:** We are developing a non-immunosuppressive treatment for GvHD that we believe will target and eliminate the T cells that cause the disease (i.e., the donor's T cells), if those T cells attack the patient's own tissues, while preserving the immune cells that are being produced by the BMT. In our GvHD product candidate, donor T cells are modified using a gene transfer vector to make them susceptible to our drug candidate, AP1903. This drug candidate may be administered if GvHD occurs, potentially killing the disease-causing donor T cells. Our product candidate uses the ARGENT cell-signaling regulation technology, our proprietary system for controlling cellular events with small molecules.

We believe that our AP1903 T cell immunotherapy product candidate can be integrated into routine BMT procedures, as follows:

- T cells would be isolated from a donor using standard procedures and be genetically engineered to introduce an inactive form of the apoptosis, or cell death, gene, known as Fas;



- The engineered donor lymphocytes would then be infused into the patient at the time of BMT or at predefined times after BMT;
- If GvHD occurs, the patient would receive our small-molecule drug, AP1903;
- AP1903 activates Fas cell signaling and would rapidly cause the donor's T cells to die, leaving the underlying bone marrow and immune system unaffected and thus potentially treating the primary cause of GvHD.

We believe that our AP1903 T cell immunotherapy product candidate will have a favorable impact on patient outcome and increase the number of patients who could benefit from allogeneic BMT by improving the risk-to-benefit ratio of the treatment. AP1903 was found to be safe and well tolerated in a Phase 1 clinical study. In addition, this study showed that AP1903 reached blood levels that are expected to be clinically effective. Our product candidate for GvHD is in phase 2 development.

## **Osteoporosis**

**The Disease:** Osteoporosis, or porous bone, is characterized by low bone mass and structural deterioration of bone tissue, leading to bone fragility and increased susceptibility to fractures, most commonly of the hip, spine and wrist. Bone is a living substance in which the tissue is constantly being broken down (resorption), while new bone is being formed. This process is called bone turnover. A full cycle of normal bone remodeling takes about two to three months. Bone resorption is accomplished by specialized cells in bone, known as osteoclasts, and new bone is generated by another group of special cells, known as osteoblasts. The balance or imbalance between the activity of the osteoclasts and osteoblasts determines whether bone mass increases, remains the same or decreases over time.

During the first 20 to 30 years of life, bone regeneration is greater than bone resorption which results in a net increase in bone mass. Beginning at about 35 years of age, a slow phase of bone resorption begins in which bone breakdown is slightly greater than bone regeneration. This slow phase continues well into old age in both men and women resulting in a net decrease in bone mass. Superimposed onto this slow phase of bone loss, women experience an accelerated postmenopausal phase of bone resorption and can cause the loss of up to 20% of their bone mass within five to seven years following menopause. According to the National Osteoporosis Foundation, osteoporosis is a major public health threat for more than 28 million Americans, 80% of whom are women. In the United States alone, ten million individuals (eight million women and two million men) are believed to already have the disease, and 18 million more have low bone mass, or osteopenia), placing them at risk for osteoporosis.

Osteoporosis is often called a "silent disease," because bone loss occurs without symptoms. People may not know that they have osteoporosis until their bones become so weak that a sudden strain, bump or fall causes a fracture or a vertebra to collapse. One in two women and one in eight men over the age of 50 are expected to have osteoporosis-related fractures. Vertebral fractures, which occur in 27% of women 65 years of age or older, may initially be felt or seen in the form of severe back pain, loss of height or spinal deformities such as a stooped posture. According to the National Osteoporosis Foundation, the cost of osteoporosis-related hip fractures alone is approaching \$14 billion annually in the U.S.

**Current Therapies:** There is no known cure for osteoporosis today. However, several medications are marketed for the prevention or treatment of osteoporosis. The major activity of these products is to reduce the bone resorbing activity of the osteoclasts, thereby reducing or preventing further bone loss. In addition, decreasing osteoclast activity provides an opportunity to indirectly shift the balance of activity to the osteoblasts which further assists in prevention of further bone breakdown along with a potential for some increase in bone mass.

Marketed treatments for osteoporosis include estrogen replacement therapy, or ERT, and Selective Estrogen Receptor Modulators, or SERMs. ERT has been available for several years and provides a beneficial effect through hormonal mechanisms. The major risk-to-benefit issue that needs to be considered in using this therapy is the association of ERT with the risk of developing cancer of the uterine lining, or endometrial cancer. The more recent availability of SERMs holds the promise of reduced risk of cancer. Additional experience, however, will be needed to clarify their comparative risks. Other reported side effects with SERMs include hot flashes and the occurrence of blood clots in deep veins.

Another marketed treatment for osteoporosis is the oral administration of a non-hormonal class of drugs known as bisphosphonates. They inhibit osteoclast-mediated bone resorption and, as such, have been widely used. These drugs are associated with side effects that can be disturbing to some patients. In particular, they are irritating to the lining of the upper gastrointestinal tract causing some patients to experience nausea, heartburn and irritation of the esophagus. Although the incidence of these side effects can be reduced by carefully controlling the administration of drug in relation to food intake, as well as controlling the physical activity of the patient for a time period following each administration, we believe that patient compliance would be greatly enhanced if more palatable, effective drugs become available.

Calcitonin, a naturally occurring non-sex hormone involved in bone metabolism, is also approved for treatment of osteoporosis. This drug is not available in an orally administered form and must be taken by nasal inhalation. Other drugs, such as parathyroid hormone administered under the skin, have undergone clinical trials and are awaiting regulatory approval.

**Our Approach:** While each of the above therapies provides beneficial effects to patients with osteoporosis, there is an opportunity to develop better-tolerated and more-effective drugs. One major advance would be the introduction of a novel class of drugs that not only inhibits osteoclast-mediated bone resorption but also directly stimulates the growth of new bone. Our scientists are developing such a class of small-molecule drug candidates for the treatment of osteoporosis and related bone diseases, including Paget's disease and periodontal disease. The drugs that are being developed are designed to interact with a specific protein in bone cells that is critically involved in bone breakdown and bone formation. Src, a protein tyrosine kinase enzyme, has been shown to be a validated target for osteoporosis through genomics experiments. In a special strain of mice that was genetically manipulated to delete this gene, several groups of researchers found that the deletion prevented bone resorption, increased bone mass and enhanced bone formation. Based on these observations, we anticipate that a drug capable of inhibiting the activity of this tyrosine kinase should provide the same effects as observed in the specific gene-depleted mice.

*In vitro* and *in vivo* animal studies have demonstrated that our lead product candidates for the treatment of osteoporosis, AP23485 and its analogs, are potent Src inhibitors that have beneficial effects on both stages of bone remodeling as predicted in the validation models. We anticipate that the dual action of these compounds will provide an extremely important advancement in treatment, prevention and potential reversal of osteoporosis. AP23485 and its analogs are in preclinical development. Further studies aimed at selecting one of the AP23485 analogs as a clinical candidate and at optimizing the formulations of these analogs for convenient dosing are underway.

### **Additional Drug Discovery Programs**

**Overview:** The regulation of cell signaling is a part of normal cellular function, and defects play critical roles in many major diseases. As a result, our technologies have a broad range of potential therapeutic applications, and we have several research programs aimed at developing additional product candidates. These programs leverage the knowledge and expertise we have gained in the development of our current lead product candidates and our focus on the hematology and oncology area.

**Cell-signaling Inhibitors that Target Cancer:** We are developing a series of highly potent small-molecule compounds that are designed to selectively inhibit oncogenic cell-signaling protein tyrosine kinases that have been implicated convincingly in human cancer and are well validated cell-signaling targets for treating cancer. Protein tyrosine kinases, such as c-Src, c-Abl, and Her2/neu, are known to play important roles in regulating tumor growth and proliferation, apoptosis (cell death), angiogenesis (blood supply), and metastasis (spread to distant sites). Using structure-based drug design and chemo-informatics, our scientists have discovered proprietary compounds that target these protein kinases and block the growth of breast, prostate, colon, lung, bone and kidney tumors and leukemia in various models. Further studies aimed at optimizing these compounds for therapeutic use are ongoing.

**Cellular Therapy:** Stem cells are “master” cells that retain the ability to specialize, or “differentiate,” into many different types of specialized cells. Recent research has emphasized the broad potential of stem cells, both embryonic and adult forms, to treat disease by providing a source of cells that can be used to replace defective cells, tissues or even whole organs. We have demonstrated, both *in vitro* and *in vivo*, that our cell-signaling regulation technologies can potentially overcome the two key limitations to the widespread use of stem-cell based therapies: (1) the inability to transfer therapeutic or corrective genes into stem cells efficiently; and (2) the subsequent difficulty in reliably deriving large numbers of specialized cells of the correct type and purity to patients.

We have also demonstrated regulated growth of other potentially useful cell types, using ARGENT cell-growth switches customized for the desired cell therapy product. These include liver cells (for the treatment of hepatic disease), muscle cells (for the treatment of heart failure), and pancreatic islet cells (for the treatment of diabetes). At present, we are evaluating these opportunities for potential product development.

**Orally Regulated Protein Therapy:** Our cell-signaling regulation technologies are a promising general platform for the delivery of secreted therapeutic proteins, because they have the potential to control the level of gene expression using an orally administered drug. Protein levels can be optimized within a therapeutic window. Allowing therapy to be terminated, if necessary, enhances safety. We believe that information obtained from the development of our Epo product candidate for anemia could be used to accelerate the development of additional protein therapy product candidates.

## **Our Core Competencies**

Our research programs are built around key areas of competency in structure-based drug design and chemo-informatics, functional genomics and proteomics, and protein engineering. The integration of these strengths provides us with unique opportunities in the era of post-genomic drug discovery.

Functional genomics and proteomics are the study of gene and protein function, or more specifically the study of how particular genes regulate cellular function. A further aspect of functional genomics is the study of how the protein products are linked in cell-signaling pathways and how these pathways are regulated. Functional genomics has particular relevance to the process of identifying specific disease-related molecular targets for drug discovery, a process termed target validation.

Protein engineering is the design and modification of proteins based on the knowledge of their atomic level structure, obtained through the use of protein X-ray crystallography or nuclear magnetic resonance spectroscopy. Usually, the design process utilizes the three-dimensional structure of the protein to incorporate non-native amino acids into the protein's structure. This process generates new surface characteristics, thereby altering the small molecule or protein binding properties of the protein.

Structure-based drug design is a computational approach used to design small organic drug molecules that bind specifically to a particular protein in a cell-signaling pathway, for example, the critical molecular target in that pathway known to be linked to a disease. Using the target protein's three-dimensional atomic

structure, drugs can be designed and optimized to bind both tightly and selectively to the target, which should lead to more potent drugs with fewer side effects. Structure-based drug design integrates structural biology and computer-assisted molecular modeling methods and has been applied directly to validated molecular targets in our cell-signaling programs to discover and optimize lead compounds. Chemo-informatic techniques and virtual screening further expand the utility of structural methods in drug discovery.

## **Our Enabling Platform Technologies**

### ***NF- $\kappa$ B Cell-signaling Technology***

Dr. David Baltimore, a founding member of our Board of Scientific and Medical Advisors, and his colleagues discovered NF- $\kappa$ B, a cellular protein that plays a critical role in cell-signaling pathways that regulate the transcription of key genes involved in many major diseases, including atherosclerosis, asthma, arthritis, cancer, diabetes, infectious disease, inflammatory bowel disease, rheumatoid arthritis, sepsis and stroke.

Recognizing the importance of NF- $\kappa$ B as a validated drug target, the pharmaceutical industry has developed and is developing a number of drugs designed to regulate the activity of this protein and other targets in its pathway. We have established what we believe to be a dominant NF- $\kappa$ B intellectual property position based on an exclusive license to the pioneering work of the Baltimore laboratory from Massachusetts Institute of Technology, The Whitehead Institute for Biomedical Research and Harvard University.

### ***Regulation Technologies to Accelerate Drug Discovery***

**Overview:** Our proprietary portfolio of cell-signaling regulation technologies includes the ARGENT Signaling, ARGENT Transcription, and RPD Secretion Technologies (collectively known as our RegTech technologies). Intracellular processes can be controlled with small molecules providing versatile tools for use in cell biology, functional genomics, proteomics, and drug-discovery research. To maximize their use by the scientific community, we distribute our technologies free of charge to academic investigators in the form of our Regulation Kits. Over 500 investigators worldwide already are using our Regulation Kits in diverse areas of research, and over 120 scientific papers describing their use have been published. For researchers in pharmaceutical and biotechnology companies, we have established an alternative licensing program to provide them with access to our cell-signaling regulation technologies on commercial terms.

**Target Validation – Cell Signaling:** As analysis of the human genome sequence uncovers a wealth of new uncharacterized genes, a key challenge will be validating those genes that are good drug targets. Many of these are likely to be signaling proteins. Our ARGENT Signaling Technology allows single signaling proteins to be activated in isolation, allowing their precise functional role to be assessed *in vitro* and then *in vivo*. ARGENT tools are effective for early analysis of newly identified “orphan” signaling proteins, because no knowledge of natural ligands or binding partners is required. In addition, identification of new pathway components and gene expression changes that occur with activation can be used to identify and further validate new drug targets.

Once a cell-signaling pathway has been validated, the same ARGENT technology can provide useful tools for the next stages of drug development. The inducible gene can be engineered into experimental animals to provide an ARGENT model of the associated disease. ARGENT cell lines in which the validated signaling complex can be inducibly activated also can provide the basis for highly targeted cell-based screening for small-molecule drug candidates.

**Target Validation – Gene Transcription:** Varying the expression level of a gene is an effective way to study its function. The tight, dose-dependent control of expression afforded by our ARGENT Transcription

Technology allows precise correlation of gene expression levels with their physiological consequences. Our technology also can be used to inducibly express inhibitors of supposed targets, such as dominant-negative mutants or gene-specific DNA binding proteins, for validation purposes.

A major application of our ARGENT Transcription Technology, based on its tight regulation of genes, is the creation of inducible knockout mice. Knockout mice in which both copies of a gene of interest have been eliminated are extremely useful for assessing the role of the deleted gene in disease. Unfortunately, many knockout mice are not viable, because expression of the gene is required during embryonic development. In addition, complete knockouts often suffer from changes in the expression of other genes that may compromise interpretation of the resulting physical, biochemical, and physiological makeup of the animal, or its phenotype. We believe that both of these problems can be solved using our ARGENT Transcription Technology by generating inducible knockouts in which genes are eliminated in the adult mouse by administering a small molecule.

**Product Validation:** The human genome sequence provides a rich source of potential proteins that are themselves drug candidates. In addition, advances in protein and antibody engineering are increasingly yielding large numbers of novel proteins that have therapeutic potential. Validating these molecules as products required extensive efforts in protein manufacturing, purification, scale-up and formulation. Inducible expression in animals can be used to validate therapeutic protein product candidates, in particular, secreted proteins and monoclonal antibodies, without the need to express and purify large amounts of recombinant protein. Since the level of protein delivered can be precisely controlled, this approach offers an effective way to characterize both the therapeutic and safety profiles of protein product candidates.

Our ARGENT Transcription and RPD Secretion Technologies provide complementary alternatives to this approach to product validation. The use of our ARGENT Transcription Technology allows a protein to be delivered over the course of several days, whereas the alternative approach based on our RPD Secretion Technology is particularly useful for generating rapid bursts of protein expression. The use of our ARGENT and RPD technologies to validate protein therapeutic candidates has particular value when a large number of related proteins need to be evaluated, as studies can be done on a high-throughput basis.

**Drug Screening:** The ability to induce a specific cell signaling, gene activation or protein secretion event in a cell allows the configuration of “targeted” cell-based screens in which the unique cell context of interest for drug design can be chemically induced. These screens very specifically search for drugs affecting cells in which a particular cell signaling or gene activation event has occurred. The tight regulation afforded by our ARGENT and RPD technologies means that highly specific screens can be set up, using the uninduced cell line as a stringent counter-screen. Because the cellular event of interest can be induced chemically, the induction step can be configured into high-throughput screens.

## **Our Business Strategy**

Our business strategy balances near-term revenues from licensing with longer-term product development. To achieve this goal, we plan to:

- Develop our current lead product candidates *at least* through phase 2 clinical trials;
- Establish the commercial infrastructure to market our hematology and oncology lead products in the United States;
- Pursue a worldwide partner for our osteoporosis product candidate and partners for our hematology and oncology lead products outside the United States, generally after we are able to obtain phase 2 clinical data;

- License our cell-signaling regulation technologies and our NF- $\kappa$ B intellectual property portfolio to biotechnology and pharmaceutical companies to accelerate their genomics, proteomics and drug discovery programs; and
- Partner our cell-signaling regulation technologies for joint development of novel products, especially with companies that have proprietary therapeutic genes, cellular systems (e.g., stem cells) or gene delivery vectors.

## **Research and Development Spending**

During each of the three years ended December 31, 2001, 2000 and 1999, respectively, we spent approximately \$16.6 million, \$12.5 million, and \$28.8 million, respectively, on our research and development activities.

## **Manufacturing**

When advantageous, we intend to rely on strategic partners or third-party contractors for manufacturing cGMP material to be used in our product candidates. We believe that our small-molecule drugs can be produced in commercial quantities through conventional synthetic and natural-product fermentation techniques. We expect to access manufacturing methods for viral vectors from potential partners and third-party manufacturers. Thus far, we have contracted with various commercial and academic entities to develop and optimize our manufacturing methods, but we have not entered into any formal manufacturing agreements adequate to produce our product candidates for large-scale clinical trials or commercial use.

## **Intellectual Property**

Patents and other intellectual property rights are essential to our business. We file patent applications to protect our technology, inventions and improvements to our inventions that are considered important to the development of our business.

As of March 18, 2002, we have 116 patents and pending patent applications in the United States, of which 54 are owned, co-owned or exclusively licensed by us and 62 are owned, co-owned or exclusively licensed by our subsidiary, ARIAD Gene Therapeutics, Inc., or AGTI. In addition, we have filed foreign counterparts, as appropriate. We also have several nonexclusive technology licenses from certain institutions in support of our research programs. We anticipate that we will continue to seek licenses from universities and others where applicable technology complements our research and development efforts.

Many of the patents and patent applications in our portfolio cover our cell-signaling regulation technologies. These patents and pending applications cover regulatory technologies, specialized variants of the technologies, critical nucleic acid components, small-molecule drugs, the identification and use of dimerizer hormone mimetics, and various uses of the technologies in health care and drug discovery. Patents issued to date include 24 patents covering our cell-signaling regulation technologies. These patents issued in the United States beginning in November 1998 and should provide proprietary protection for our protein and cell therapy product candidates until at least 2015. We hope to obtain additional patents in the ensuing years based on pending applications.

Our patent portfolio also covers research tools and methods used in our drug discovery programs, as well as multiple classes of small-molecule compounds discovered in those programs. We also have a number of issued patents and pending applications relating to the cell-signaling proteins, NF- $\kappa$ B and NF-AT, and their use in drug discovery.

We also rely on unpatented trade secrets and proprietary know-how. However, trade secrets are difficult to protect. We enter into confidentiality agreements with our employees, consultants and collaborators. In addition, we believe that certain technologies utilized in our research and development programs are in the public domain. Accordingly, we do not believe that patent or other protection is available for these technologies. If a third party were to obtain patent or other proprietary protection for any of these technologies, we may be required to challenge such protections, obtain a license for such technologies or terminate or modify our programs that rely on such technologies.

### **Our Board of Scientific and Medical Advisors**

We have assembled a Board of Scientific and Medical Advisors that currently consist of experts in the fields of molecular and cellular biology, biochemistry, immunology, and organic, physical, and computational chemistry, and molecular medicine. Each advisor is engaged under a consulting agreement that requires the advisor to provide consulting services to us in our field of interest and not to disclose any of our confidential information. Our Board of Scientific and Medical Advisors is chaired by Dr. Stuart L. Schreiber, Morris Loeb Professor and Chair, Chemistry and Chemical Biology; Co-Director, Institute of Chemistry and Cell Biology; and Scientific Co-Director, Bauer Center of Genomics Research at Harvard University and an Investigator of the Howard Hughes Medical Institute.

### **Our Licenses**

We and our subsidiary, AGTI, have entered into license agreements with various research institutions and universities pursuant to which we and/or AGTI are the licensee of certain technologies upon which some of our product candidates are based.

We have agreed to pay royalties to our licensors on sales of certain products based on the licensed technologies, as well as, in some instances, milestone payments and patent filing and prosecution costs. The licenses also impose various milestones, commercialization, sublicensing, royalty as well as insurance and other obligations. Failure by us to comply with these requirements could result in the termination of the applicable agreement, which could have a material adverse effect on our business, financial condition, and results of operations.

### **Competition**

In the area of cell-signaling regulation, companies such as Amgen, Inc., AstraZeneca plc, Biogen, Inc., Eli Lilly and Co., Imclone Systems, Inc., Ligand Pharmaceuticals, Inc., Millennium Pharmaceuticals, Inc., Novartis Pharma AG, OSI Pharmaceuticals, Inc., Tularik, Inc., and Vertex Pharmaceuticals, Inc. are developing drugs to treat human disease by inhibiting cell-signaling pathways. Companies developing gene-delivery technologies related to our programs include Avigen, Inc., Biogen, Inc., Cell Genesys, Inc., Genzyme Corp., MediGene, GmbH, and Targeted Genetics Corp. Several companies are developing products to treat GvHD, including Abgenix, Inc., AVAX, Inc., BioTransplant, Inc., Protein Design Labs, Inc., and Repligen Corp. Other companies have products on the market or in development against which our products may have to compete. In the area of oncology, such companies include Wyeth Corp., Bristol-Myers Squibb Co., Glaxo Smith Kline plc., and Pharmacia, Inc. In the field of bone diseases, such companies include Merck & Co., Inc., NPS Pharmaceuticals, Inc., and Proctor and Gamble, Inc. We may also experience competition from companies that have acquired or may acquire technology from companies, universities, and other research institutions. As these companies develop their technologies, they may develop proprietary positions that may materially and adversely affect us.

## Government Regulation

Our ongoing research and development activities, our clinical trials, the manufacturing and testing procedures and the marketing of our product candidates, if they are approved, all are subject to extensive regulation by numerous governmental authorities in the United States and other countries. Any drug developed by us must undergo rigorous preclinical studies and clinical testing and an extensive regulatory approval process administered by the FDA under the federal Food, Drug and Cosmetic Act prior to marketing in the United States. Satisfaction of such regulatory requirements, which includes demonstrating that a product is both safe and effective for its intended indications for use, typically takes several years or more depending upon the type, complexity and novelty of the product and requires the expenditure of substantial resources. Preclinical studies must be conducted in conformance with the FDA's good laboratory practice regulations. Before commencing clinical trials in the United States, we must submit to and receive clearance from the FDA of an IND. There can be no assurance that submission of an IND will result in FDA clearance to commence clinical trials. Clinical testing must meet requirements for IRB oversight, informed consent and good clinical practice and is subject to continuing FDA oversight. In addition, certain clinical studies involving gene transfer conducted in the United States require the review and approval of the National Institutes of Health Recombinant DNA Advisory Committee, or the RAC. There can be no assurance that submission to the RAC will result in clearance to commence clinical trials. We have a limited history of conducting preclinical studies and the clinical trials necessary to obtain regulatory approval. Furthermore, we or the FDA may suspend clinical trials at any time if either party believes that the subjects participating in such trials are being exposed to unacceptable risks or if the FDA finds deficiencies in the conduct of the trials or other problems with our product under development.

Before receiving FDA approval to market a product, we will have to demonstrate that the product is safe and effective in the patient population that will be treated. Data obtained from preclinical studies and clinical trials are susceptible to varying interpretations that could delay, limit or prevent regulatory clearances. In addition, delays or rejections may be encountered based upon additional government regulation from future legislation or administrative action or changes in FDA policy during the period of product development, clinical trials and FDA regulatory review. Similar delays also may be encountered in foreign countries. There can be no assurance that even after such time and expenditures, regulatory approval will be obtained for any product candidates developed by us, or, even if approval is obtained, that the labeling for such products will not limit the product's condition of use, which could materially impact the marketability and profitability of the product. If regulatory approval of a product is granted, such approval will be limited to those disease states and conditions for which the product has been shown useful, as demonstrated by clinical trials. Furthermore, approval may entail ongoing requirements for post-market studies. Even if such regulatory approval is obtained, a marketed product, its manufacturer and its manufacturing facilities and procedures are subject to continual review and periodic inspections by the FDA. Discovery of previously unknown problems with a product, manufacturer, manufacturing procedures or facility may result in restrictions on such product or manufacturer, including costly recalls, an injunction against continued marketing and manufacturing until the problems have been adequately addressed to the FDA's satisfaction or even withdrawal of the product from the market. There can be no assurance that any compound developed by us alone or in conjunction with others will prove to be safe and efficacious in clinical trials and will meet all of the applicable regulatory requirements needed to receive and maintain marketing approval. Additionally, the marketing, labeling and advertising for an approved product is subject to ongoing FDA scrutiny and the failure to adhere to applicable requirements can result in regulatory action that could have a material adverse impact on the profitability of the product.

Outside the United States, our ability to market a product will be contingent upon receiving a marketing authorization from the appropriate regulatory authorities. The requirements governing the conduct of clinical trials, marketing authorization, pricing and reimbursement vary widely from country to country. At present, foreign marketing authorizations are applied for at a national level, although within the European Community certain registration procedures are available to companies wishing to market a product in more



than one member state. If the regulatory authority is satisfied that adequate evidence of safety, quality and efficacy has been presented, a marketing authorization will be granted. This foreign regulatory approval process includes all of the risks associated with FDA clearance set forth above.

## **Our Employees**

As of March 18, 2002, we had 69 employees, 34 of whom hold post-graduate degrees, including 18 with a Ph.D. or M.D. Most of our employees are engaged directly in research and development. We have entered into confidentiality and noncompetition agreements with all of our employees. None of our employees are covered by a collective bargaining agreement, and we consider relations with our employees to be good.

## **RISK FACTORS**

***THE RISKS AND UNCERTAINTIES DESCRIBED BELOW ARE THOSE THAT WE CURRENTLY BELIEVE MAY MATERIALLY AFFECT OUR COMPANY. ADDITIONAL RISKS AND UNCERTAINTIES THAT WE ARE UNAWARE OF OR THAT WE CURRENTLY DEEM IMMATERIAL ALSO MAY BECOME IMPORTANT FACTORS THAT AFFECT OUR COMPANY.***

### **Risks Relating to Our Business**

***We may never succeed in developing marketable drugs or generating product revenues.***

We are an early-stage company with no product revenues, and we may not succeed in producing pharmaceutical products for commercialization. We do not expect to have any products on the market for several years, if at all. Our main focus is research and product development. We are exploring human diseases at the cellular level. We seek to discover which genes within cells malfunction to cause disease, which signals are triggered within cells during the disease process to cause these cells to respond abnormally and which drugs can halt or reverse those activities within cells. As with all science, we face much trial and error, and we may fail at numerous stages along the way. If we are not successful in developing marketable products, we will not be profitable.

***We have incurred significant losses to date and may never be profitable.***

We have incurred significant operating losses in each year since our formation in 1991 as a Delaware corporation through 2001 and have an accumulated deficit of approximately \$108.5 million from our operations through December 31, 2001. It is likely that we will incur significant operating losses for the foreseeable future. We currently have no product revenues or commitments for future research revenues, may never be able to earn such revenues and may never have profitable operations, even if we are able to commercialize any of our product candidates or enter into additional research agreements. If our losses continue and we are unable to successfully develop, commercialize, manufacture and market product candidates, we may never have product revenues or achieve profitability. Losses have resulted principally from costs incurred in research and development of product candidates and from general and administrative costs associated with our operations.

***Insufficient funding may jeopardize our research and development programs and may prevent commercialization of our products and technologies.***

All of our operating revenue to date has been generated through collaborative research agreements that have expired or been terminated. Accordingly, we may not be able to secure the significant funding which is required to maintain and continue each of our research and development programs at the current levels or at levels that may be required in the future. We do not have any committed strategic alliance funding for the advancement of any of our programs. Although we intend to seek additional funding from product-based

collaborations, technology licensing, and public and private financings, additional funding may not be available on terms acceptable to us, or at all. If we cannot secure adequate financing, we may be required to delay, scale back or eliminate one or more of our research and development programs or to enter into license arrangements with third parties to commercialize products or technologies that we would otherwise seek to develop ourselves.

***Because we do not own all of the outstanding stock of our subsidiary, ARIAD Gene Therapeutics, Inc., or AGTI, we may not realize all of the potential future economic benefit from products developed based on technology licensed to or owned by our subsidiary.***

Our subsidiary, AGTI, holds licenses from Harvard University, Stanford University and other universities relating to our ARGENT cell-signaling regulation technology, a key component of our small-molecule regulated anemia and graft-vs-host disease product candidates. Minority stockholders of AGTI, including Harvard University, Stanford University, some of our scientific advisors, and some current and former members of our management, own 20% of the issued and outstanding capital stock of AGTI. We own the remaining 80% of the issued and outstanding capital stock of AGTI. We do not currently have a license agreement with AGTI that provides us with rights to commercialize products based on our ARGENT cell-signaling regulation technology or products based on technology or compounds derived from our ARGENT programs. In order to commercialize any product based on these technologies or compounds, we will either license them on terms to be determined or commercialize these products through AGTI. The economic benefit to our stockholders from products that we commercialize will be diminished by any royalties paid under a future license agreement, if any, with AGTI. The economic benefit to our stockholders from products, if any, that AGTI may commercialize would be reduced in an amount related to the percentage owned by the minority stockholders of AGTI.

Alternatively, we may acquire all of the interests of the minority stockholders in AGTI for cash, shares of our common stock or other securities of ours, if any. AGTI has a right of first refusal on the sale to third parties of 73% of the minority stockholders' AGTI shares. AGTI does not have a call option, or a right to require the minority stockholders to sell their shares to us, for any of these shares. If we acquire these minority interests for either form of consideration, it will result in dilution to our stockholders. The economic value of the minority stockholders' interests is difficult to quantify in the absence of a public market, and the market price of our publicly traded common stock may not accurately reflect its value. Accordingly, the market could change its perception of the value of these minority interests in our subsidiary at any time in reaction to our increased emphasis on these product candidates, announcements regarding these product candidates or for other reasons, any of which could result in a decline in our stock price. In addition, if we acquire the minority interests at a cost greater than the value attributed to them by the market, this also could result in a decline in our stock price. If we choose to acquire these minority interests through a short-form merger in which we do not solicit the consent of the minority stockholders of AGTI, we could become subject to an appraisal procedure, which would result in additional expense and diversion of management resources.

***Because members of our management team and/or board of directors beneficially own a material percentage of the capital stock of our subsidiary, AGTI, and we have agreements with AGTI, there may be conflicts of interest present in dealings between ARIAD and AGTI.***

Four members of our management team and/or board of directors own or have the right to acquire up to approximately 6.1% of the outstanding capital stock of AGTI. Harvey J. Berger, M.D., our Chairman, Chief Executive Officer and President, owns 3.4%; David L. Bernstein, Esq., our Senior Vice President and Chief Patent Counsel, owns 0.3%; John D. Iuliucci, Ph.D., our Senior Vice President, Drug Development, owns 0.7%; and Jay R. LaMarche, one of our Directors and a part-time employee, owns 1.7%. These same individuals beneficially own approximately 7.1% of our outstanding common stock. In addition, as part of the formation of AGTI, we entered into agreements with AGTI to provide for the operations of AGTI. As a result, the market may perceive conflicts of interest to exist in dealings between AGTI and us. AGTI is the

exclusive licensee of the ARGENT intellectual property from Harvard University and Stanford University and of related technologies from other universities. In the event that we commercialize products based on or derived from our ARGENT cell-signaling regulation technology or related technologies or compounds, we will have to negotiate the terms of a license agreement with AGTI or acquire all of the capital stock of AGTI that we do not currently own. Because of the apparent conflicts of interest, the market may be more inclined to perceive the terms of any transaction between us and AGTI as being unfair to us.

*The loss of key members of our scientific and management staff could delay and may prevent the achievement of our research, development and business objectives.*

Our Chairman, Chief Executive Officer and President, Harvey J. Berger, M.D.; our Senior Vice President and Chief Patent Counsel, David L. Berstein, Esq.; our Senior Vice President, Drug Development, John D. Iuliucci, Ph.D.; our Senior Vice President and Chief Business Officer, Fritz Casselman; and other key officers and members of our scientific staff responsible for areas such as drug development, regulatory affairs, drug discovery, and manufacturing are important to our specialized scientific business. We also are dependent upon a few of our scientific advisors to assist in formulating our research and development strategy. The loss of, and failure to promptly replace, any member of our management team could significantly delay and may prevent the achievement of our research, development and business objectives. While we have entered into employment agreements with all of our officers, these officers may not remain with us.

*We may be unable to develop or commercialize our product candidates, if we are unable to obtain or maintain certain licenses.*

We have entered into license agreements for some of our technologies, either directly or through AGTI. We are currently attempting to obtain additional licenses for technology useful to our programs. Our inability to obtain any one or more of these licenses, on commercially reasonable terms, or at all, or to circumvent the need for any such license, could cause significant delays and cost increases and materially affect our ability to develop and commercialize our product candidates. We also use gene sequences or proteins encoded by those sequences and other biological materials in each of our research programs which are, or may become, patented by others and to which we would be required to obtain licenses in order to develop or market our product candidates. Some of our programs, including, for example, our regulated protein therapy program, may require the use of multiple proprietary technologies, especially gene-transfer vectors and therapeutic genes. Obtaining licenses for these technologies may require us to make cumulative royalty payments or other payments to several third parties, potentially reducing amounts paid to us or making the cost of our products commercially prohibitive.

Some of our licenses obligate us to exercise diligence in pursuing the development of product candidates, to make specified milestone payments and to pay royalties. In some instances, we are responsible for the costs of filing and prosecuting patent applications. These licenses generally expire upon the earlier of a fixed term of years after the date of the license or the expiration of the applicable patents, but each license is also terminable by the other party upon default by us of our obligations. Our inability or failure to meet our diligence requirements or make any payments required under these licenses would result in a reversion to the licensor of the rights granted which, with respect to the licenses pursuant to which we have obtained exclusive rights, would materially and adversely affect our ability to develop and market products based on our licensed technologies.

*We may be unable to access vectors or other gene transfer technologies that we will need to develop and commercialize our regulated protein and cellular therapy product candidates.*

We may not be able to access the gene transfer technologies required to develop, manufacture, and commercialize our regulated protein and cellular therapy product candidates. We are reliant on our ability to enter into license agreements with appropriate academic institutions and/or gene therapy companies that

can provide us with rights to the necessary technology, production methods, and components of gene delivery systems. The inability to reach an appropriate agreement with such an entity on reasonable commercial terms could delay or prevent the preclinical evaluation, clinical testing and/or commercialization of our product candidates. Our inability to access gene transfer technology, including suitable manufacturing methods, would have significant adverse effects on some of our product candidates. If we do not market our product candidates, we will never become profitable. In addition, the intellectual property landscape covering gene transfer technologies is currently uncertain and fragmented. Accordingly, if we select one partner as a source for selected intellectual property rights, we may find that we have not licensed sufficient rights to be able to commercialize our products or we may be forced to acquire additional rights or discontinue marketing our product candidates unexpectedly.

*We have no experience in manufacturing any of our product candidates, which raises uncertainty as to our ability to develop and commercialize our product candidates.*

We have no experience in, and currently lack the resources and capability to, manufacture any of our product candidates on a large scale. Our ability to conduct clinical trials and commercialize our product candidates will depend, in part, on our ability to manufacture our products on a large scale, either directly or through third parties, at a competitive cost and in accordance with cGMP and other regulatory requirements. We currently do not have the capacity to manufacture our product candidates in large quantities. We depend on third-party manufacturers or collaborative partners for the production of our product candidates for preclinical research and clinical trials and intend to use third-party manufacturers to produce any products we may eventually commercialize. If we are not able to obtain contract manufacturing on commercially reasonable terms, we may not be able to conduct or complete clinical trials or commercialize our product candidates, and we do not know whether we will be able to develop such capabilities. If we are not able to develop cell processing methods that comply with recently adopted regulatory guidelines known as current Good Tissue Practices, or cGTP, we may not be able to commercialize our regulated cellular therapy products.

*Competing technologies may render some or all of our programs or future products noncompetitive or obsolete.*

Many well-known pharmaceutical, healthcare and biotechnology companies, academic and research institutions and government agencies, which have substantially greater capital, research and development capabilities and experience than us, are presently engaged in:

- Developing products based on cell signaling, genomics, proteomics, computational chemistry and protein and cellular therapies; and
- Conducting research and development programs for the treatment of all the disease areas in which we are focused.

Some of these entities already have product candidates in clinical trials or in more advanced preclinical studies than we do. These entities may succeed in commercializing competitive products before us, which would give them a competitive advantage. Competing technologies may render some or all of our programs or future products noncompetitive or obsolete, and we may not be able to make the enhancements to our technology necessary to compete successfully with newly emerging technologies. If we are unable to compete in our chosen markets, we will not become profitable.

*We may not be able to protect our intellectual proprietary rights.*

We and our licensors have pending patent applications covering biochemical and cellular tests useful in drug discovery, new chemical compounds discovered in our drug discovery programs, certain components, configurations and uses of our cell-signaling regulation technologies, and methods and materials for conducting pharmaceutical research. These patent applications may not issue as patents and may not issue in all countries in which we develop, manufacture or sell our products. In addition, patents issued to us or our licensors may be challenged and subsequently narrowed, invalidated or circumvented. In that event, such patents may not afford meaningful protection for our technologies or product candidates, which would materially impact our ability to develop and market our product candidates. Certain technologies utilized in our research and development programs are already in the public domain. Moreover, a number of our competitors have developed technologies, filed patent applications or obtained patents on technologies and compositions that are related to our business and may cover or conflict with our patent applications. Such conflicts could limit the scope of the patents that we may be able to obtain or may result in the denial of our patent applications. If a third party were to obtain intellectual proprietary protection for any of these technologies, we may be required to challenge such protections, terminate or modify our programs that rely on such technologies or obtain licenses for use of these technologies.

*If our product candidates are not accepted by physicians and insurers, we will not be successful.*

Our success is dependent on the acceptance of our product candidates. Our product candidates may not achieve significant market acceptance among patients, physicians or third-party payors, even if we obtain necessary regulatory and reimbursement approvals. Failure to achieve significant market acceptance of our product candidates will harm our business. We believe that recommendations by physicians and health care payors will be essential for market acceptance of any product candidates. In the past, there has been concern regarding the potential safety and effectiveness of gene therapy products. Physicians and health care payors may conclude that any of our product candidates are not safe.

*If we are unable to establish sales, marketing and distribution capabilities or to enter into agreements with third parties to do so, we may be unable to successfully market and sell any products.*

We currently have no sales, marketing or distribution capabilities. If we are unable to establish sales, marketing or distribution capabilities either by developing our own sales, marketing and distribution organization or by entering into agreements with others, we may be unable to successfully sell any products that we are able to begin to commercialize. If we are unable to effectively sell our products, our ability to generate revenues will be harmed. We may not be able to hire, in a timely manner, the qualified sales and marketing personnel we need, if at all. In addition, we may not be able to enter into any marketing or distribution agreements on acceptable terms, if at all. If we cannot establish sales, marketing and distribution capabilities as we intend, either by developing our own capabilities or entering into agreements with third parties, sales of future products, if any, may be harmed.

*If we develop a product for commercial use, a subsequent product liability-related claim or recall could have an adverse effect on our business.*

Our business exposes us to potential product liability risks inherent in the testing, manufacturing and marketing of pharmaceutical products, and we may not be able to avoid significant product liability exposure. A product liability-related claim or recall could be detrimental to our business. In addition, except for insurance covering product use in our clinical trials, we do not currently have any product liability insurance, and we may not be able to obtain or maintain such insurance on acceptable terms, or we may not be able to obtain any insurance to provide adequate coverage against potential liabilities. Our inability to obtain sufficient insurance coverage at an acceptable cost or otherwise to protect against potential product liability claims could prevent or limit the commercialization of any products that we develop.

## **Risks Relating to Governmental Approvals**

*We have limited experience in conducting clinical trials, which may cause delays in commencing and completing clinical trials of our product candidates.*

Clinical trials must meet FDA and foreign regulatory requirements. We have limited experience in conducting the preclinical studies and clinical trials necessary to obtain regulatory approval. Consequently, we may encounter problems in clinical trials that may cause us or the FDA or foreign regulatory agencies to delay, suspend or terminate these trials. If the clinical trials of our product candidates fail, we will not be able to market our product candidates. Problems we may encounter include the possibility that we may not be able to manufacture sufficient quantities of cGMP materials for use in clinical trials, conduct clinical trials at preferred sites, enroll sufficient test subjects or begin or successfully complete clinical trials in a timely fashion, if at all. Furthermore, we, the FDA or foreign regulatory agencies may suspend clinical trials at any time if we or they believe the subjects participating in the trials are being exposed to unacceptable health risks or if we or they find deficiencies in the clinical trial process or conduct of the investigation. The FDA and foreign regulatory agencies could also require additional clinical trials, which would result in increased costs and significant development delays. Our failure to adequately demonstrate the safety and effectiveness of a therapeutic product candidate under development could delay or prevent regulatory approval of the product candidate and could have a material adverse effect on our business.

*We may not be able to obtain government regulatory approval for our product candidates prior to marketing.*

To date, we have not submitted a marketing application for any product candidate to the FDA or any foreign regulatory agency, and none of our product candidates have been approved for commercialization in the United States or elsewhere. Prior to commercialization, each product candidate would be subject to an extensive and lengthy governmental regulatory approval process in the United States and in other countries. We may not be able to obtain regulatory approval for any product candidate we develop or even if approval is obtained, the labeling for such products may be required to bear limitations that could materially impact the marketability and profitability of the product involved. We have no history of conducting and managing the clinical testing necessary to obtain such regulatory approval. Satisfaction of these regulatory requirements, which includes satisfying the FDA and foreign regulatory authorities that the product is both safe and effective under its intended indications of use, typically takes several years or more depending upon the type, complexity and novelty of the product and requires the expenditure of substantial resources.

Furthermore, the regulatory requirements governing our potential products are uncertain. This uncertainty may result in excessive costs or extensive delays in the regulatory approval process, adding to the already lengthy review process. If regulatory approval of a product is granted, such approval will be limited to those disease states and conditions for which the product is proven safe and effective, as demonstrated by clinical trials, and our products will be subject to ongoing regulatory reviews. Although we have been granted orphan drug designation by the FDA for AP1903, the small-molecule drug used in our GvHD product candidate, this designation may be challenged by others or may prove to be of no practical benefit.

*We will not be able to sell our product candidates, if we or our third-party manufacturers fail to comply with FDA manufacturing regulations.*

Before we can begin to commercially manufacture our product candidates, we must either secure manufacturing in an approved manufacturing facility or obtain regulatory approval of our own manufacturing facility and processes. In addition, the manufacturing of our product candidates must comply with the FDA's cGMP and/or cGTP requirements. These requirements govern, among other things, quality control and documentation procedures. We, or any third-party manufacturer of our product candidates, may not be able to comply with these requirements, which would prevent us from selling such

products. Material changes to the manufacturing processes of our products after approvals have been granted are also subject to review and approval by the FDA or other regulatory agencies.

*Even if we bring products to market, we may be unable to effectively price our products or obtain adequate reimbursement for sales of our products, which would prevent our products from becoming profitable.*

If we succeed in bringing our product candidates to the market, they may not be considered cost-effective, and reimbursement to the patient may not be available or may not be sufficient to allow us to sell our products on a competitive basis. In both the United States and elsewhere, sales of medical products and treatments are dependent, in part, on the availability of reimbursement to the patient from third-party payors, such as government and private insurance plans. Third-party payors are increasingly challenging the prices charged for pharmaceutical products and services. Our business is affected by the efforts of government and third-party payors to contain or reduce the cost of health care through various means. In the United States, there have been and will continue to be a number of federal and state proposals to implement government controls on pricing. In addition, the emphasis on managed care in the United States has increased and will continue to increase the pressure on the pricing of pharmaceutical products. We cannot predict whether any legislative or regulatory proposals will be adopted or the effect these proposals or managed care efforts may have on our business.

### **Risks Relating to Our Common Stock**

*Results of our operations and general market conditions for biotechnology stocks could result in the sudden change in the value of our stock.*

As a biopharmaceutical company, we have experienced significant volatility in our common stock. Fluctuations in our operating results and general market conditions for biotechnology stocks could have a significant impact on the volatility of our common stock price. During 2001, our stock price ranged from a high of \$8.38 to a low of \$1.66. Factors contributing to such volatility include: results and timing of preclinical studies and clinical trials; evidence of the safety or effectiveness of pharmaceutical products; announcements of new collaborations; failure to enter into collaborations; our funding requirements; announcements of technological innovations or new therapeutic products; governmental regulation; policies regarding recombinant DNA and gene therapy; healthcare or cost-containment legislation; developments in patent or other proprietary rights, including litigation; general market trends for the biotechnology industry and related high-technology industries; the impact of changing interest rates and policies of the Federal Reserve; and public policy pronouncements.

### **ITEM 2: PROPERTIES**

We have leased approximately 100,000 square feet (approximately 37,000 square feet currently under sublease to third parties) of laboratory and office space at 26 Landsdowne Street, located at University Park at M.I.T., in Cambridge, Massachusetts. The lease is for a ten-year term ending in July of 2002, with two consecutive five-year renewal options. We have extended the lease for the first five-year option. We believe that our currently leased facility will, in large part, be adequate for our research and development activities at least through the year 2004.

### **ITEM 3: LEGAL PROCEEDINGS**

We were named as a defendant in a purported class action lawsuit commenced in June 1995 in the U.S. District Court for the Southern District of New York. The action named as defendants ARIAD Pharmaceuticals, Inc.; the underwriter of our initial public offering and a market maker in our stock, D. Blech & Co.; the managing director and sole shareholder of D. Blech & Co. and one of our former directors, David

Blech; certain other of our directors, and the qualified independent underwriter for the initial public offering, Shoenberg Hieber, Inc., or SHI.

Counsel for the plaintiff class, counsel for the Company and the named director defendants, excluding David Blech, or the Company Defendants, and counsel for SHI have executed a stipulation of settlement in the action, or the Proposed Settlement. The Proposed Settlement, in substance, contemplates a payment of \$620,000 as consideration for plaintiffs' consent to entry of judgment dismissing the action with prejudice and barring "contribution-type" claims against the Company Defendants by non-settling parties. The Proposed Settlement further is subject to the Court's approval of that stipulation as fair, adequate and reasonable, and to entry of an appropriate judgment of dismissal in the action and in a related action entitled In re: Blech Securities Litigation, 94 Civ. 7696 (RWS), from which the Court previously ordered us dismissed as a defendant. The amount we have contributed to a legal escrow account was not material, and accordingly, no liability is recorded on the 2001 balance sheet.

On May 19, 1999, we filed suit in the Massachusetts Superior Court against Michael Z. Gilman, Ph.D., or Dr. Gilman, our former Chief Scientific Officer, seeking equitable relief for breach of his employment agreements in accepting a position as the research director of molecular biology at Biogen, Inc., or Biogen. The Superior Court issued a temporary injunction on May 19, 1999 restraining Dr. Gilman from using any of our confidential information in his new employment. On June 21, 1999, Dr. Gilman filed counterclaims against us seeking an order awarding damages for breach of contract and barring us from enforcing any provisions of our employment agreements with Dr. Gilman. On May 26, 1999 Biogen filed a motion to intervene as a defendant in the action which the Superior Court granted on August 2, 1999. Discovery in the case has been completed, and Summary Judgment Motions have been filed, heard and ruled upon. Our claim against Dr. Gilman and Dr. Gilman's counterclaims will now proceed to trial. No trial date has been set. The ultimate outcome of the litigation with Dr. Gilman is not determinable or estimatable at this time, and accordingly, no liability is recorded on the balance sheets.

#### **ITEM 4: SUBMISSION OF MATTERS TO A VOTE OF SECURITY HOLDERS**

No matters were submitted to a vote of security holders during the quarter ended December 31, 2001.



## PART II

### ITEM 5: MARKET FOR REGISTRANT'S COMMON EQUITY AND RELATED STOCKHOLDER MATTERS

#### Market Information

Our common stock has been traded on the Nasdaq National Market under the symbol "ARIA" since September 19, 1994. The following table sets forth the high and low sales prices of our common stock as quoted on the Nasdaq National Market for the periods indicated.

<b>2001:</b>	<b>High</b>	<b>Low</b>
First Quarter	\$ 8.38	\$ 2.78
Second Quarter	6.84	3.80
Third Quarter	5.40	1.66
Fourth Quarter	6.40	2.41
<b>2000:</b>		
First Quarter	\$ 48.50	\$ 2.50
Second Quarter	17.50	5.69
Third Quarter	15.88	8.31
Fourth Quarter	13.00	4.50

#### Holders

The approximate number of holders of record of our common stock as of March 18, 2002 was 461, and the approximate total number of beneficial holders of our common stock as of March 18, 2002 was \_\_TBD\_\_.

#### Dividends

We have not declared or paid dividends on our common stock in the past and do not intend to declare or pay such dividends in the foreseeable future. Our long-term debt agreement prohibits the payment of cash dividends. (See "Management's Discussion and Analysis of Financial Condition and Results of Operations - Liquidity and Capital Resources" and Note 5 of "Notes to Consolidated Financial Statements.")

## ITEM 6: SELECTED FINANCIAL DATA

The selected financial data set forth below as of December 31, 2001, 2000, 1999, 1998 and 1997 and for the years then ended have been derived from the audited consolidated financial statements of the Company, of which the financial statements as of December 31, 2001 and 2000 and for the years ended December 31, 2001, 2000, and 1999 are included elsewhere in this Annual Report on Form 10-K and are qualified by reference to such financial statements. The information set forth below should be read in conjunction with "Management's Discussion and Analysis of Financial Condition and Results of Operations" and the audited consolidated financial statements, and the notes thereto, and other financial information included herein.

<i>In thousands, except share and per share data</i>	Years Ended December 31,				
	2001	2000	1999	1998	1997
<b>Consolidated Statements of Operations Data:</b>					
Research revenue (principally related parties prior to 2000) \$	4	\$ 128	\$ 12,468	\$ 12,143	\$ 9,234
Operating expenses:					
Research and development	16,587	12,467	28,844	35,515	20,287
General and administrative	4,469	3,318	3,938	2,634	2,925
Total operating expenses	<u>21,056</u>	<u>15,785</u>	<u>32,782</u>	<u>38,149</u>	<u>23,212</u>
Loss from operations	(21,052)	(15,657)	(20,314)	(26,006)	(13,978)
Other income (expense):					
Interest income	1,578	2,050	445	999	1,757
Interest expense	(285)	(225)	(522)	(481)	(410)
Gain on sale of Genomics Center			46,440		
Equity in net loss of Genomics Center			(1,493)	(660)	
Total other income (expense)	<u>1,293</u>	<u>1,825</u>	<u>44,870</u>	<u>(142)</u>	<u>1,347</u>
Income (loss) before cumulative effect of change in accounting principle	(19,759)	(13,832)	24,556	(26,148)	(12,631)
Cumulative effect of change in accounting principle			(364)		
Net income (loss)	(19,759)	(13,832)	24,192	(26,148)	(12,631)
Repurchase and accretion costs attributable to redeemable convertible preferred stock			(6,435)	(36)	
Net income (loss) attributable to common stockholders	<u>\$ (19,759)</u>	<u>\$ (13,832)</u>	<u>\$ 17,757</u>	<u>\$ (26,184)</u>	<u>\$ (12,631)</u>
<b>Earnings (loss) per share:</b>					
Per common share (basic):					
Income (loss) attributable to common stockholders before cumulative effect of change in accounting principle	\$ (.68)	\$ (.53)	\$ .82	\$ (1.25)	\$ (.66)
Cumulative effect of change in accounting principle			(.02)		
Net income (loss) – basic	<u>\$ (.68)</u>	<u>\$ (.53)</u>	<u>\$ .80</u>	<u>\$ (1.25)</u>	<u>\$ (.66)</u>
Weighted average number of shares of common stock outstanding – basic	29,256,767	25,875,663	22,004,646	20,966,586	19,252,885
Per common share (diluted):					
Income (loss) before cumulative effect of change in accounting principle	\$ (.68)	\$ (.53)	\$ .71	\$ (1.25)	\$ (.66)
Cumulative effect of change in accounting principle			(.01)		
Net income (loss) – diluted	<u>\$ (.68)</u>	<u>\$ (.53)</u>	<u>\$ .70</u>	<u>\$ (1.25)</u>	<u>\$ (.66)</u>
Weighted average number of shares of common stock outstanding – diluted	29,256,767	25,875,663	34,448,015	20,966,586	19,252,885

<i>In thousands</i>	Years Ended December 31,				
	2001	2000	1999	1998	1997
<b>Consolidated Balance Sheet Data:</b>					
Cash, cash equivalents and marketable securities	\$ 47,186	\$ 39,781	\$ 28,320	\$ 14,176	\$ 29,359
Working capital	42,775	36,866	22,731	5,806	16,539
Total assets	55,361	48,813	44,236	30,786	47,409
Long-term debt	6,847	3,700	1,900	3,295	5,156
Redeemable convertible preferred stock			8,070	5,036	
Accumulated deficit	(108,474)	(88,715)	(74,883)	(92,640)	(66,457)
Stockholders' equity	43,093	40,851	27,068	11,733	28,374

## ITEM 7: MANAGEMENT'S DISCUSSION AND ANALYSIS OF FINANCIAL CONDITION AND RESULTS OF OPERATIONS

*You should read the following discussion and analysis in conjunction with "Selected Consolidated Financial Data" and our consolidated financial statements and the related notes included elsewhere in this report.*

### Overview

We are engaged in the discovery and development of breakthrough medicines that regulate cell signaling with small molecules. Breakthrough medicines are products, created *de novo*, that may be used to treat diseases in innovative ways. Our lead product candidates – treatments for cancer, cancer that has spread to bone, or bone metastases, anemia, graft-vs-host disease following T cell immunotherapy and osteoporosis – all were developed through the integration of genomics, proteomics and structure-based drug design. We have an exclusive license to pioneering technology related to the discovery, development, and use of drugs that modulate the cellular protein, NF- $\kappa$ B, and other targets in its pathway, which regulate key genes involved in many major diseases.

### Aventis Relationship

From November 1995 through December 1999, substantially all of our research revenue and the majority of our research expenses were incurred in collaboration with Aventis Pharmaceuticals Inc., or Aventis (formerly known as Hoechst Marion Roussel, Inc.) and its affiliates.

In November 1995, we entered into an agreement with Hoechst Marion Roussel, S.A. to collaborate on the discovery and development of drugs to treat osteoporosis and related bone diseases, or the Osteoporosis Agreement, one of our cell-signaling inhibitor programs. In March 1997, we entered into an agreement, which established a 50%/50% joint venture, called the Hoechst-ARIAD Genomics Center, LLC, or the Genomics Center, with Aventis to pursue functional genomics with the goal of identifying genes that encode novel therapeutic proteins and small-molecule drug targets. We recognized aggregate revenue under these agreements of \$12.5 million in 1999, \$11.7 million in 1998 and \$8.7 million in 1997.

On December 31, 1999, we completed the sale of our 50% interest in the Genomics Center to Aventis, and as a result: (1) we received \$40.0 million in cash; (2) 3,004,436 shares of our series B preferred stock were returned to us; (3) Aventis forgave \$1.9 million of long-term debt we owed to them; (4) we received drug candidates and related technologies resulting from the Osteoporosis Agreement; and (5) we received the right to use certain genomics and bioinformatics technologies developed by the Genomics Center. We recorded a net gain on the sale of \$46.4 million. As a result of this sale, we did not receive any revenue from our relationship with Aventis after 1999, and we realized a reduction of revenue in fiscal 2000 of \$12.4 million from 1999, which was more than offset by a reduction in research and development expenses of approximately \$16.3 million, primarily associated with the Genomics Center.

### General

Since our inception in 1991, we have devoted substantially all of our resources to our research and development programs. We receive no revenue from the sale of pharmaceutical products, and substantially all revenue to date has been received in connection with our past relationship with Aventis. Except for the gain on the sale of the Genomics Center in December 1999, which resulted in net income for fiscal 1999, we have not been profitable since inception. We expect to incur substantial and increasing operating losses for the foreseeable future, primarily due to the expansion of our pharmaceutical product development programs, clinical trials, and product manufacturing. We expect that losses will fluctuate from quarter to quarter and that these fluctuations may be substantial. As of December 31, 2001, we had an accumulated deficit of \$108.5 million.

Our business plan aims to balance near-term revenues from licensing with longer-term product development. To achieve this goal, we plan to develop our lead product candidates at least through phase 2 clinical trials, establish the commercial infrastructure to market our hematology and oncology lead products in the United States, pursue a worldwide partner for our osteoporosis product candidate and partners for our hematology and oncology lead products outside the United States, generally after we are able to obtain phase 2 clinical data, license our cell-signaling regulation technologies and our NF- $\kappa$ B intellectual property portfolio to biotechnology and pharmaceutical companies to accelerate their genomics, proteomics and drug discovery programs, and partner our cell-signaling regulation technologies for joint development of novel products, especially with companies that have proprietary therapeutic genes, cellular systems (e.g., stem cells) or gene delivery vectors. However, there can be no assurance that we will be successful in achieving our strategies and generating future revenue streams. As of January 1, 2002, we had no collaborative agreements that would generate revenue in 2002.

### **Critical Accounting Policies**

Our financial position and results of operations are affected by subjective and complex judgments, particularly in the areas of stock-based compensation to consultants and the carrying value of intangible assets. In determining stock-based compensation expense, we utilize a financial model that takes into account, among other things, the price and volatility of our common stock, an interest-free discount rate, and an estimate of the life of the option contract. Fluctuations in those factors result in uneven expense charges to our statement of operations.

At December 31, 2001, we reported \$5.3 million of intangible assets consisting of costs related primarily to purchased patents, patent applications and licenses. These costs are being amortized over the estimated useful lives of the underlying intangible assets. Changes in these lives or a decision to discontinue using the technologies could result in material changes to our balance sheet and statements of operations.

### **Results of Operations**

#### ***Years Ended December 31, 2001 and 2000***

#### **Revenue**

We recognized research revenue of \$4,000 for the year ended December 31, 2001 compared to \$128,000 for the year ended December 31, 2000. The decrease in research revenue was due to the termination of our services agreements with the Genomics Center as a result of the sale of our 50% ownership interest in the Genomics Center to Aventis on December 31, 1999. Research revenue for the year ended December 31, 2000 was comprised principally of transitional research revenue for services provided to Aventis following the December 31, 1999 sale of our interest in the Genomics Center. As of January 1, 2002, we had no collaborative agreements that would generate revenue in 2002.

#### **Operating expenses**

Research and development expenses increased 33% to \$16.6 million in 2001 from \$12.5 million in 2000. This \$4.1 million increase in 2001 expenses as compared to those incurred in 2000 was primarily due to a higher level of spending on product development of \$867,000, product manufacturing and external activities in support of clinical trials of \$1.7 million, costs associated with the launch of our initiatives to promote the commercialization and licensing of our cell-signaling regulation technologies by both corporate and academic researchers of \$325,000, increased personnel expenses of \$567,000 and overhead expenses of \$471,000. We expect our research and development expenses to increase over the next year as a result of our continued expansion of our product development programs, clinical trials and product manufacturing. However, the amount of such increase in research and development spending will be determined, in part, by

our ability to attract additional capital or to realize revenues through partnerships, licensing, joint ventures, or similar arrangements.

General and administrative expenses increased 35% to \$4.5 million in 2001 from \$3.3 million in 2000. This \$1.2 million increase in 2001 expenses as compared to those incurred in 2000 was primarily due to increased professional expenses of \$419,000 and personnel expenses of \$825,000.

#### **Other income/expense**

Interest income decreased 23% to \$1.6 million in 2001 from \$2.1 million in 2000 primarily as a result of declining interest rates during the year. Interest expense increased 27% to \$285,000 in 2001 from \$225,000 in 2000. This increase was primarily due to a higher level of long-term debt outstanding in 2001.

#### **Operating Results**

We reported a loss from operations of \$21.1 million in 2001 compared to a loss from operations of \$15.7 million in 2000, an increase in loss of \$5.4 million or 34%. We expect that operating losses will increase and be substantial for several more years as our product development activities expand, and these losses are expected to fluctuate as a result of differences in the timing and composition of revenue earned and expenses incurred.

We reported a net loss attributable to common stockholders of \$19.8 million in 2001 or \$.68 per share (basic and diluted). We reported a net loss attributable to common stockholders of \$13.8 million in 2000 or \$.53 per share (basic and diluted).

#### ***Years Ended December 31, 2000 and 1999***

##### **Revenue**

We recognized research revenue under our services agreements and collaborative research arrangements of \$128,000 for the year ended December 31, 2000 compared to \$12.5 million for the year ended December 31, 1999. This decrease of \$12.4 million was due to the reduction in revenues associated with the termination of our services agreements with the Genomics Center of \$6.4 million as a result of the sale of our 50% ownership interest in the Genomics Center and the termination of the Osteoporosis Agreement of \$6.0 million.

##### **Operating Expenses**

Research and development expenses decreased 57% to \$12.5 million in 2000 from \$28.8 million in 1999. This decrease of \$16.3 million in 2000 expenses as compared to those incurred in 1999 was primarily due to the termination of research services provided to the Genomics Center.

General and administrative expenses decreased 16% to \$3.3 million in 2000 from \$3.9 million in 1999. This \$620,000 decrease in 2000 expenses as compared to those incurred in 1999 was primarily due to lower professional and legal expenses.

##### **Other income/expense**

Interest income increased 361% to \$2.1 million in 2000 from \$445,000 in 1999 as a result of a higher level of invested funds. Interest expense decreased 57% to \$225,000 in 2000 from \$522,000 in 1999. This decrease was primarily due to a lower level of debt outstanding in 2000 as a result of repayments on our borrowings.

## Accounting Change

We adopted Statement of Position, or SOP 98-5, Reporting the Cost of Start-Up Activities, effective January 1, 1999 and recorded a charge of \$364,000 as a cumulative effect of change in accounting principle.

## Operating Results

We reported a loss from operations of \$15.7 million in 2000 compared to a loss from operations of \$20.3 million in 1999, a decrease in loss of \$4.6 million or 23%. We reported a loss of \$13.8 million in 2000 and reported income before cumulative effect of change in accounting principle of \$24.6 million in 1999. After such cumulative effect, we reported net income of \$24.2 million for 1999. Our results for 1999 included the gain on the sale of our 50% interest in the Genomics Center of \$46.4 million. Although we earned taxable income in 1999 due to the gain on the sale of the Genomics Center, we were able to utilize net operating loss carryforwards to eliminate substantially all taxes due.

On December 31, 1999 and January 14, 2000, we repurchased and retired all of our series C preferred stock and recorded a charge of \$6.2 million in 1999 representing the premium paid on the repurchase, which has been deducted from net income in determining net income attributable to common stockholders. Accretion costs attributable to the series C preferred stock of \$250,000 were also recognized in 1999. We reported a net loss attributable to common stockholders of \$13.8 million in 2000 or \$.53 per share (basic and diluted). We reported net income attributable to common stockholders of \$17.8 million in 1999 or \$.80 per share (basic) and \$.70 per share (diluted).

## Selected Quarterly Financial Data

Summarized quarterly financial data are as follows:

*In thousands, except per share amounts*

	2001 Quarters			
	First	Second	Third	Fourth
Total research revenues	\$ 1	\$ 1	\$ 1	\$ 1
Net loss	(4,157)	(4,716)	(4,745)	(6,141)
Net loss per share - diluted	(.15)	(.17)	(.16)	(.20)

	2000 Quarters			
	First	Second	Third	Fourth
Total research revenues	\$ 108	\$ 18	\$ 1	\$ 1
Net loss	(3,522)	(3,190)	(3,287)	(3,833)
Net loss per share - diluted	(.15)	(.12)	(.12)	(.14)

## Liquidity and Capital Resources

We have financed our operations and investments primarily through the private placement and public offering of our equity securities, and research revenue and other transactions with Aventis, including the sale of our 50% interest in the Genomics Center in December 1999. In addition, we have financed our operations through the issuance of long-term debt, operating and capital-lease transactions, interest income, and government sponsored research grants.

At December 31, 2001, we had cash, cash equivalents and marketable securities totaling \$47.2 million and working capital of \$42.8 million compared to cash, cash equivalents and marketable securities totaling \$39.8 million and working capital of \$36.9 million at December 31, 2000.

The primary uses of cash during the year ended December 31, 2001 were \$16.5 million to finance our operations and working capital requirements, \$7.6 million to acquire marketable securities, \$614,000 to purchase laboratory equipment, \$1.0 million to acquire intellectual property and \$1.2 million to repay long-term debt.

The primary sources of funds during the year ended December 31, 2001 were \$34.4 million from the sales and maturities of marketable securities, \$4.6 million of new borrowings as a result of increasing our existing bank term loan (\$3.8 million) and entering into a new term loan with an equipment financing company (\$790,000), \$21.6 million from the sale of common stock and \$489,000 from the sale of common stock from the exercise of stock options and purchases under the terms of the employee stock purchase plan.

On June 22, 2001, we filed a shelf registration statement with the SEC for the issuance of up to 4.5 million registered shares of our common stock, which was declared effective by the SEC on August 1, 2001. The registered shares are available for sale at our discretion. On October 31, 2001, we sold 1,927,712 registered shares of our common stock to institutional investors at a price of \$4.15 per share and received gross proceeds of \$8.0 million before commissions and expenses of \$464,000. This net amount of \$7.5 million was included in the 2001 primary sources of funds mentioned in the preceding paragraph. At December 31, 2001, we had 2,572,288 registered shares remaining available for sale under this shelf registration. On January 9, 2002, we filed an additional shelf registration statement with the SEC for the issuance of up to 3.0 million registered shares of our common stock, which was declared effective on February 13, 2002. The registered shares are available for sale at our discretion.

We have substantial fixed contractual obligations under various research and licensing agreements, consulting and employment agreements, lease agreements and long-term debt instruments. These contractual obligations were comprised of the following as of December 31, 2001:

<i>In thousands</i>		<b>Payments Due By Period</b>			
<b>Contractual Obligations</b>	<b>Total</b>	<b>Less than 1 year</b>	<b>1 - 3 years</b>	<b>4 - 5 years</b>	<b>6th year</b>
Long-term debt	\$ 8,290	\$ 1,443	\$ 6,847	\$ --	\$ --
Operating leases	5,196	1,520	2,431	1,245	
Other long-term obligations *	<u>5,490</u>	<u>3,087</u>	<u>1,917</u>	<u>324</u>	<u>162</u>
Total fixed contractual obligations	<u>\$ 18,976</u>	<u>\$ 6,050</u>	<u>\$ 11,195</u>	<u>\$ 1,569</u>	<u>\$ 162</u>

\* Other long-term obligations are comprised primarily of employment agreements and licensing agreements.

We will require substantial additional funding for our research and development programs, including preclinical development and clinical trials, for operating expenses, for the pursuit of regulatory approvals and for establishing manufacturing, marketing and sales capabilities. Adequate funds for these purposes, whether obtained through financial markets or other arrangements with collaborative partners, or from other sources, may not be available when needed or on terms acceptable to us.

Based on the historical spending levels to support our operations, our available funds will be adequate to satisfy our capital and operating requirements for the next two years. However, there can be no assurance



that changes in our research and development plans or other future events affecting our revenues or operating expenses will not result in the earlier depletion of our funds.

At December 31, 2001, we had available for federal tax reporting purposes net operating loss carryforwards of approximately \$110.1 million that expire commencing in 2009 and for state tax reporting purposes, net operating loss carryforwards of approximately \$85.2 million that expire commencing in 2002. We also had federal research and development tax credit carryovers of approximately \$6.0 million that expire commencing in 2006. The utilization of both the net operating loss carryforwards and tax credits is subject to certain limitations under federal tax laws.

### **New Accounting Pronouncements**

In June 1998, the Financial Accounting Standards Board issued SFAS No. 133, *Accounting for Derivative Instruments and Hedging Activities*. The new standard, which was adopted on January 1, 2001, requires that all companies record derivatives on the balance sheet as assets or liabilities, measured at fair value. Gains or losses resulting from changes in the values of those derivatives are accounted for depending on the use of the derivative and whether it qualifies for hedge accounting. The adoption of this standard on January 1, 2001 had no impact on our financial position or results of operations.

In June 2001, the Financial Accounting Standards Board issued SFAS No. 141, *Business Combinations*, and SFAS No. 142, *Goodwill and Other Intangible Assets*. SFAS No. 141 supersedes APB No. 16, *Business Combinations*, and SFAS No. 38, *Accounting for Preacquisition Contingencies of Purchased Enterprises*, and requires that all business combinations be accounted for by a single method – the purchase method. SFAS No. 141 also provides guidance on the recognition of intangible assets identified in a business combination and requires enhanced financial statement disclosures. SFAS No. 142 adopts a more aggregate view of goodwill and bases the accounting for goodwill on the units of the combined entity into which an acquired entity is integrated. In addition, SFAS No. 142 concludes that goodwill and intangible assets that have indefinite useful lives will not be amortized but rather will be tested at least annually for impairment. Intangible assets that have finite lives will continue to be amortized over their useful lives. SFAS No. 141 is effective for all business combinations initiated after June 30, 2001. The adoption of SFAS No. 142 is required for fiscal years beginning after December 15, 2001 (the year 2002 for the Company), except for the nonamortization and amortization provisions which are required for goodwill and intangible assets acquired after June 30, 2001. We believe that the adoption of SFAS No. 141 and SFAS No. 142 will not have a material impact on our financial position or results of operation.

In October 2001, the Financial Accounting Standards Board issued SFAS No. 144, “Accounting for the Impairment or Disposal of Long-Lived Assets.” SFAS No. 144 supersedes previous guidelines for financial accounting and reporting for the impairment or disposal of long-lived assets and for segments of a business to be disposed of. We believe that the adoption of SFAS No. 144 will not have a material impact on our financial position or results of operations.

### **ITEM 7A: QUANTITATIVE AND QUALITATIVE DISCLOSURES ABOUT MARKET RISK**

We maintain an investment portfolio in accordance with our investment policy to preserve principal, maintain proper liquidity to meet operating needs and maximize yields. Our investment policy specifies credit quality standards for our investments and limits the amount of credit exposure to any single issue, issuer or type of investment.

We invest cash balances in excess of operating requirements in short-term securities, generally with maturities of 90 days or less. Our marketable securities generally consist of corporate debt and U.S. Government securities primarily with maturities of one year or less, but generally less than six months. These securities are classified as available-for-sale. Available-for-sale securities are recorded on the balance sheet at fair market value with unrealized gains or losses reported as a separate component of stockholders'

equity (accumulated other comprehensive loss). Gains and losses on marketable security transactions are reported on the specific-identification method. Interest income is recognized when earned. A decline in the market value of any available-for-sale security below cost that is deemed other than temporary results in a charge to earnings and establishes a new cost basis for the security. These investments are sensitive to interest rate risk. We believe that the effect, if any, of reasonable possible near-term changes in the interest rates on our financial position, results of operations and cash flows would not be material due to the short-term nature of these investments.

We have an executive compensation plan which provides a deferred compensation benefit for certain executives and key employees. Under the plan, benefits are deferred and generally vest over four years. The benefits obligation is recorded as compensation and a liability based on the underlying fair market value of the obligation as it vests. As of December 31, 2001, in the event of a hypothetical 10% increase in the underlying fair market value of the obligation, we would incur approximately \$47,000 of additional compensation expense in 2002.

At December 31, 2001, we have a bank term note which bears interest at prime plus 1%. This note is sensitive to interest rate risk. In the event of a hypothetical 10% increase in the prime rate (47.5 basis points), we would incur approximately \$33,000 of additional interest expense per year.

### **Certain Factors That May Affect Future Results of Operations**

The SEC encourages companies to disclose forward-looking information so that investors can better understand a company's future prospects and make informed investment decisions. This Annual Report contains such "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. These statements may be made directly in this Annual Report, and they may also be made a part of this Annual Report by reference to other documents filed with the SEC, which is known as "incorporation by reference."

Words such as "may," "anticipate," "estimate," "expects," "projects," "intends," "plans," "believes" and words and terms of similar substance used in connection with any discussion of future operating or financial performance, identify forward-looking statements. All forward-looking statements are management's present expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially from those described in the forward-looking statements. These risks include, but are not limited to, risks and uncertainties regarding the Company's preclinical studies, the Company's ability to conduct clinical trials of its product candidates and the results of such trials, as well as risks and uncertainties relating to economic conditions, markets, products, competition, intellectual property, services and prices, key employees, future capital needs, dependence on the Company's collaborators and other factors. Please also see the discussion of risks and uncertainties under "Risk Factors" in Item 1 of this Report.

In light of these assumptions, risks and uncertainties, the results and events discussed in the forward-looking statements contained in this Annual Report or in any document incorporated by reference might not occur. Stockholders are cautioned not to place undue reliance on the forward-looking statements, which speak only of the date of this Annual Report or the date of the document incorporated by reference in this Annual Report. We are not under any obligation, and we expressly disclaim any obligation, to update or alter any forward-looking statements, whether as a result of new information, future events or otherwise. All subsequent forward-looking statements attributable to the Company or to any person acting on its behalf are expressly qualified in their entirety by the cautionary statements contained or referred to in this section.

## ITEM 8: FINANCIAL STATEMENTS AND SUPPLEMENTARY DATA

### Independent Auditors' Report

The Board of Directors and Stockholders of  
ARIAD Pharmaceuticals, Inc.:

We have audited the accompanying consolidated balance sheets of ARIAD Pharmaceuticals, Inc. and its subsidiaries (the "Company") as of December 31, 2001 and 2000, and the related consolidated statements of operations, stockholders' equity, and cash flows for each of the three years in the period ended December 31, 2001. These financial statements are the responsibility of the Company's management. Our responsibility is to express an opinion on these consolidated financial statements based on our audits.

We conducted our audits in accordance with auditing standards generally accepted in the United States of America. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audits provide a reasonable basis for our opinion.

In our opinion, such consolidated financial statements present fairly, in all material respects, the financial position of ARIAD Pharmaceuticals, Inc. and its subsidiaries as of December 31, 2001 and 2000, and the results of their operations and their cash flows for each of the three years in the period ended December 31, 2001, in conformity with accounting principles generally accepted in the United States of America.

As discussed in Note 1 to the financial statements, in 1999 the Company changed its method of accounting for start-up activities.

/s/DELOITTE & TOUCHE LLP

Boston, Massachusetts  
January 18, 2002

**ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES**  
**CONSOLIDATED BALANCE SHEETS**

<i>In thousands, except share and per share data</i>	December 31,	
	<u>2001</u>	<u>2000</u>
<b>ASSETS</b>		
Current assets:		
Cash and cash equivalents	\$ 46,742	\$ 12,543
Marketable securities	444	27,238
Inventory and other current assets	1,010	1,347
Total current assets	<u>48,196</u>	<u>41,128</u>
Property and equipment:		
Leasehold improvements	12,624	12,606
Equipment and furniture	5,417	4,821
Total	18,041	17,427
Less accumulated depreciation and amortization	<u>(16,190)</u>	<u>(14,914)</u>
Property and equipment, net	<u>1,851</u>	<u>2,513</u>
Intangible and other assets, net	5,314	5,172
Total assets	<u><u>\$ 55,361</u></u>	<u><u>\$ 48,813</u></u>
 <b>LIABILITIES AND STOCKHOLDERS' EQUITY</b>		
Current liabilities:		
Accounts payable	\$ 1,505	\$ 1,434
Current portion of long-term debt	1,443	1,200
Accrued compensation and benefits	822	540
Accrued product developments expenses	1,073	460
Accrued expenses	578	628
Total current liabilities	<u>5,421</u>	<u>4,262</u>
Long-term debt	<u>6,847</u>	<u>3,700</u>
Commitments, contingent liabilities and minority interest (Notes 1, 6, 10)		
Stockholders' equity:		
Preferred stock, authorized, 10,000,000 shares, none issued and outstanding		
Common stock, \$.001 par value, authorized, 60,000,000 shares, issued and outstanding, 32,146,774 shares in 2001 and 27,292,138 shares in 2000	32	27
Additional paid-in capital	151,638	129,761
Deferred compensation	(106)	(217)
Accumulated other comprehensive income (loss)	3	(5)
Accumulated deficit	<u>(108,474)</u>	<u>(88,715)</u>
Total stockholders' equity	<u>43,093</u>	<u>40,851</u>
Total liabilities and stockholders' equity	<u><u>\$ 55,361</u></u>	<u><u>\$ 48,813</u></u>

See notes to consolidated financial statements.

**ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES**  
**CONSOLIDATED STATEMENTS OF OPERATIONS**

	<b>Years Ended December 31,</b>		
<i>In thousands, except share and per share data</i>	<u><b>2001</b></u>	<u><b>2000</b></u>	<u><b>1999</b></u>
Research revenue (principally related parties in 1999)	\$ 4	\$ 128	\$ 12,468
Operating expenses:			
Research and development *	16,587	12,467	28,844
General and administrative *	<u>4,469</u>	<u>3,318</u>	<u>3,938</u>
Total operating expenses	<u>21,056</u>	<u>15,785</u>	<u>32,782</u>
Loss from operations	(21,052)	(15,657)	(20,314)
Other income (expense):			
Interest income	1,578	2,050	445
Interest expense	(285)	(225)	(522)
Gain on sale of Genomics Center			46,440
Equity in net loss of Genomics Center			<u>(1,493)</u>
Total other income	<u>1,293</u>	<u>1,825</u>	<u>44,870</u>
Income (loss) before cumulative effect of change in accounting principle	(19,759)	(13,832)	24,556
Cumulative effect of change in accounting principle			<u>(364)</u>
Net income (loss)	(19,759)	(13,832)	24,192
Repurchase and accretion costs attributable to redeemable convertible preferred stock			<u>(6,435)</u>
Net income (loss) attributable to common stockholders	<u>\$ (19,759)</u>	<u>\$ (13,832)</u>	<u>\$ 17,757</u>
Earnings (loss) per share:			
Per common share (basic):			
Income (loss) attributable to common stockholders before cumulative effect of change in accounting principle	\$ (.68)	\$ (.53 )	\$ .82
Cumulative effect of change in accounting principle			<u>(.02)</u>
Net income (loss) – basic	<u>\$ (.68)</u>	<u>\$ (.53)</u>	<u>\$ .80</u>
Weighted average number of shares of common stock outstanding – basic	29,256,767	25,875,663	22,004,646
Per common share (diluted):			
Income (loss) before cumulative effect of change in accounting principle	\$ (.68)	\$ (.53 )	\$ .71
Cumulative effect of change in accounting principle			<u>(.01)</u>
Net income (loss) – diluted	<u>\$ (.68)</u>	<u>\$ (.53)</u>	<u>\$ .70</u>
Weighted average number of shares of common stock outstanding – diluted	29,256,767	25,875,663	34,448,015
*Includes non-cash stock-based compensation expense			
- Research and development expense	\$ 146	\$ 142	\$ 86
- General and administrative expense	\$ 57		

See notes to consolidated financial statements.

**ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES**  
**CONSOLIDATED STATEMENTS OF STOCKHOLDERS' EQUITY**  
**For the Years Ended December 31, 1999, 2000 and 2001**

In thousands, except share data	Series B Convertible Preferred Stock		Common Stock		Additional Paid-in Capital		Accumulated Other Comprehensive Income (Loss)		Stockholders' Equity	
	Shares	Amount	Shares	Amount			Deferred Compensation	Accumulated Deficit		
Balance, January 1, 1999	2,526,316	\$ 25	21,938,754	\$ 22	\$ 104,361	\$ 0	\$ (34)	\$ (92,640)	\$ 11,734	
Additional issuance of Series B Convertible Preferred Stock	478,120	5			5,742				5,742	
Issuance of shares pursuant to ARIAD and AGTI stock option plans and an ARIAD stock purchase plan			93,134		159				159	
Stock-based compensation to consultants					86				86	
Repurchase and accretion costs attributable to series C preferred stock								(6,435)	(6,435)	
Redemption on sale of Genomics Center	(3,004,436)	(30)			(8,420)				(8,450)	
Comprehensive income:										
Net income								24,192	24,192	
Other comprehensive income -							34		34	
Unrealized gains on marketable securities										
Comprehensive income										34
Balance, December 31, 1999	0	0	22,031,888	22	101,928	0	0	(74,883)	27,067	
Issuance of common stock, series C settlement			1,078,038	1	1,144				1,145	
Issuance of common stock, net of issuance costs			857,024	1	9,742				9,743	
Exercise of warrants			1,389,498	1	11,636				11,637	
Issuance of shares pursuant to ARIAD and AGTI stock option plans and an ARIAD stock purchase plan			1,935,690	2	4,952				4,954	
Stock-based compensation to consultants					359		(359)			
Amortization of stock-based compensation							142		142	
Comprehensive loss:										
Net loss								(13,832)	(13,832)	
Other comprehensive loss-										
Unrealized loss on marketable securities							(5)		(5)	
Comprehensive loss										
Balance, December 31, 2000	0	0	27,292,138	27	129,761	(217)	(5)	(88,715)	40,851	
Issuance of common stock, net of issuance costs			4,551,541	5	21,296				21,301	
Issuance of shares pursuant to ARIAD stock option and purchase plans			303,095		489				489	
Stock-based compensation to consultants					92		(92)			
Amortization of stock-based compensation							203		203	
Comprehensive loss:										
Net loss								(19,759)	(19,759)	
Other comprehensive income -							8		8	
Unrealized gains on marketable securities										
Comprehensive loss										
Balance, December 31, 2001	0	0	32,146,774	32	\$ 151,638	\$ (106)	\$ 3	\$ (108,474)	\$ 43,093	

See notes to consolidated financial statements

**ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES**  
**CONSOLIDATED STATEMENTS OF CASH FLOWS**

<i>In thousands</i>	Years Ended December 31,		
	2001	2000	1999
Cash flows from operating activities:			
Net income (loss)	\$ (19,759)	\$ (13,832)	\$ 24,192
Adjustments to reconcile net income (loss) to net cash used in operating activities:			
Depreciation and amortization	1,889	1,675	3,682
Deferred compensation	175	163	102
Stock-based compensation to consultants	203	142	86
Gain on sale of the Genomics Center			(46,440)
Increase (decrease) from:			
Inventory and other current assets	337	262	584
Due from Genomics Center			333
Other assets	(53)	(408)	72
Accounts payable	70	(842)	(1,046)
Accrued compensation and benefits	282	58	(91)
Accrued product development expenses	438	103	(680)
Accrued expenses	(50)	(2,393)	619
Advance from Genomics Center		(26)	(3,137)
Net cash used in operating activities	<u>(16,468)</u>	<u>(15,098)</u>	<u>(21,724)</u>
Cash flows from investing activities:			
Acquisitions of marketable securities	(7,585)	(42,965)	(211)
Proceeds from sales and maturities of marketable securities	34,356	15,805	7,806
Investment in property and equipment, net	(614)	(447)	(677)
Acquisition of intangible and other assets	(1,002)	(1,205)	(710)
Proceeds from disposition of investment in Genomics Center			40,000
Investment in Genomics Center			(6,261)
Return of investment in Genomics Center			7,960
Net cash provided by (used in) investing activities	<u>25,155</u>	<u>(28,812)</u>	<u>47,907</u>

See notes to consolidated financial statements.

(Continued)

**ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES**  
**CONSOLIDATED STATEMENTS OF CASH FLOWS**

<i>In thousands</i>	Years Ended December 31,		
	2001	2000	1999
Cash flows from financing activities:			
Proceeds from long-term debt borrowings	\$ 4,590	\$ 3,000	
Repayment of long-term debt borrowings	(1,200)	(1,200)	\$ (2,056)
Proceeds from exercise of warrants, net		11,637	
Proceeds from sale/leaseback of equipment, net			309
Proceeds from issuance of common stock, net of issuance costs	21,633	9,743	
Proceeds from issuance of common stock pursuant to stock option and purchase plans	489	4,953	159
Proceeds from issuance of series B convertible preferred stock			5,747
Proceeds from related party long-term debt			1,801
Repurchase of redeemable convertible preferred stock			(10,325)
Net cash provided by (used in) financing activities	<u>25,512</u>	<u>28,133</u>	<u>(4,365)</u>
Net increase (decrease) in cash and equivalents	34,199	(15,777)	21,818
Cash and equivalents, beginning of year	<u>12,543</u>	<u>28,320</u>	<u>6,502</u>
Cash and equivalents, end of year	<u>\$ 46,742</u>	<u>\$ 12,543</u>	<u>\$ 28,320</u>

See notes to consolidated financial statements.

(Concluded)



## ARIAD PHARMACEUTICALS, INC. AND SUBSIDIARIES

### NOTES TO CONSOLIDATED FINANCIAL STATEMENTS

#### 1. Nature of Business and Summary of Significant Accounting Policies

##### *Nature of Business*

The Company is engaged in the discovery and development of breakthrough medicines that regulate cell signaling with small molecules. Breakthrough medicines are products, created *de novo*, that may be used to treat diseases in innovative ways. The Company's lead product candidates – treatment of cancer, cancer that has spread to the bone, or bone metastases, anemia, graft-vs-host disease following T cell immunotherapy and osteoporosis – all were developed through the integration of genomics, proteomics and structure-based drug design. The Company has an exclusive license to pioneering technology related to the discovery, development and use of drugs that modulate the cellular protein, NF-κB, and other targets in its pathway, which regulate key genes involved in many major diseases.

##### *Principles of Consolidation*

The consolidated financial statements include the accounts of ARIAD Pharmaceuticals, Inc., its wholly owned subsidiary, ARIAD Corporation, and its 80% owned subsidiary (79% on a fully diluted basis with respect to AGTI), ARIAD Gene Therapeutics, Inc. ("AGTI") (Note 8). The Company's research and development relating to product candidates based on its ARGENT cell-signaling regulation technology and product candidates based on technology or compounds derived from its ARGENT programs are conducted by AGTI. Intercompany accounts and transactions have been eliminated. There is no minority interest for AGTI recorded in the consolidated balance sheet, because AGTI currently has a deficiency in its stockholders' equity. The Company accounts for any gain from the exercise of AGTI options as an adjustment to additional paid-in capital. Because AGTI is a research and development company, the Company believes that the gain realization from the exercise of AGTI options cannot be assured and therefore should be accounted for as a capital transaction in the Company's consolidated financial statements.

##### *Fair Value of Financial Instruments*

The carrying amounts of cash, cash equivalents, accounts payable and accrued liabilities approximate fair value because of their short-term nature. Marketable securities are recorded in the consolidated financial statements at aggregate fair value (Note 2). The carrying amount of the Company's bank term note approximate fair value due to the variable interest rate (Note 5). The fair value of the General Electric Capital Corporation term note was \$790,000 as of December 31, 2001.

##### *Accounting Estimates*

The preparation of the consolidated financial statements in conformity with accounting principles generally accepted in the United States of America requires management to make estimates and assumptions that affect the reported amounts and disclosure of assets and liabilities at the date of the consolidated financial statements and the reported amounts and disclosure of revenue and expenses during the reporting period. Actual results could differ from those estimates.

##### *Cash Equivalents*

Cash equivalents include short-term, highly liquid investments, which consist principally of United States Treasury and Agency securities, high-grade domestic corporate securities, purchased with remaining maturities of 90 days or less and money market accounts.

### *Marketable Securities*

The Company has classified its marketable securities as “available-for-sale” and, accordingly, carries such securities at aggregate fair value. The difference between fair value and original cost is reflected as a component of accumulated other comprehensive income (loss). Fair value has been determined based on quoted market prices, in a dealer market, at the closing bid for each individual security held.

### *Inventory*

Inventories are carried at cost using the first-in, first-out method and are charged to research and development expense when consumed. Inventory consists of bulk pharmaceutical material to be used for multiple preclinical and clinical development programs and amounted to \$682,000 and \$898,000 at December 31, 2001 and 2000, respectively.

### *Property and Equipment*

Property and equipment are recorded at cost. Depreciation is recorded using the straight-line method over the estimated useful lives of the assets (3 to 10 years). Assets acquired under capital lease obligations are stated at the lower of the present value of the minimum lease payments or the fair market value at the inception of the lease. Assets recorded under capital leases and leasehold improvements are amortized over the shorter of their useful lives or lease term using the straight-line method (4 to 10 years).

### *Investment in Genomics Center*

The Company accounted for its investment in the Genomics Center using the equity method through December 31, 1999. Intercompany transactions were eliminated to the extent of the Company’s interest (50%) in the Genomics Center (Note 4).

### *Intangible and Other Assets*

Intangible and other assets consist primarily of purchased patents, patent applications, licenses, and deposits. The cost of purchased patents and patent applications and costs incurred in filing patents are capitalized. Capitalized costs related to patent applications are expensed, when it becomes determinable that such applications will not be pursued. Capitalized costs related to issued patents are amortized over a period not to exceed seventeen years or the remaining life of the patent, whichever is shorter, using the straight-line method. Amortization expense for intangible and other assets amounted to \$502,000, \$431,000, and \$501,000 for 2001, 2000 and 1999, respectively. Accumulated amortization of intangible and other assets at December 31, 2001 and 2000 was \$2.9 million and \$2.0 million, respectively.

### *Impairment of Long-Lived Assets*

The Company reviews its long-lived assets for impairment when events or changes in circumstances indicate that the carrying amount of a long-lived asset may not be recoverable. Recoverability of assets to be held and used is measured by a comparison of the carrying amount of an asset to future net cash flows expected to be generated by the asset. If such assets are considered to be impaired, the impairment to be recognized is measured by the amount by which the carrying amount of the assets exceeds the fair value of the assets.

### *Revenue Recognition*

Under collaborative research and development agreements, research revenue is recognized as the research is performed under the terms of the applicable agreement. Amounts received in advance under the 1995 Osteoporosis Agreement (Note 3) were recorded as deferred revenue and were amortized over the minimum term of the Agreement using the straight-line method. Revenue earned upon the attainment of research or product development milestones was recognized when achieved. Research revenue associated with the

Services Agreements with the Genomics Center (Note 4) was billed on a cost reimbursement basis, which includes direct costs incurred in connection with research activities and an allocation of certain other costs incurred by the Company and was recognized as services were provided.

### *Segment Reporting*

The Company organizes itself into one segment reporting to the chief executive officer. No significant revenues from product sales or services occurred in 2001 or 2000. In years prior to 2000, revenues were primarily derived from research and development activities with collaborative and strategic partners in the pharmaceutical industry.

### *Stock-Based Compensation*

SFAS No. 123, *Accounting for Stock-Based Compensation*, addresses the financial accounting and reporting standards for stock or other equity-based compensation arrangements. The Company has elected to continue to use the intrinsic value-based method to account for employee stock option awards under the provisions of Accounting Principles Board Opinion No. 25 and provides disclosures based on the fair value method in the notes to the financial statements as permitted by SFAS No. 123. Stock or other equity-based compensation for non-employees must be accounted for under the fair value-based method as required by SFAS No. 123 and Emerging Issues Task Force ("EITF") No. 96-18, *Accounting for Equity Instruments That Are Issued to Other Than Employees for Acquiring, or in Conjunction with Selling, Goods or Services* and other related interpretations. Under this method, the equity-based instrument is valued at either the fair value of the consideration received or the equity instrument issued on the date of grant. The resulting compensation cost is recognized and charged to operations over the service period, which is usually the vesting period.

### *Earnings Per Share*

Basic earnings per common share are computed using the weighted average number of common shares outstanding during each year. Diluted earnings per common share reflect the effect of the Company's outstanding options, warrants and convertible securities, except where such items would be anti-dilutive. In years in which a net loss is reported, basic and diluted per share amounts are the same. In 2001 and 2000, options amounting to 4,639,782, and 3,480,360 shares of common stock, respectively, were not included in the computation of dilutive earnings per share, because this effect would be anti-dilutive. In 1999, options and warrants amounting to 3,504,188 shares of common stock were not included in the computation of dilutive earnings because this effect would be anti-dilutive.

The following is a reconciliation of the shares used in the calculation of basic and diluted net income per share for the year ended December 31, 1999. Potentially dilutive shares were calculated using the treasury stock method:

	<b>1999</b>
Weighted average shares for basic shares outstanding	22,004,646
Incremental shares from assumed conversion of preferred stock	11,846,541
Incremental shares from assumed exercise of potentially dilutive stock options	596,828
Weighted average shares for dilutive shares outstanding	<u>34,448,015</u>
<b><i>In thousands</i></b>	
Net income attributable to common stockholders	\$ 17,757
Effect of repurchase and accretion costs attributable to redeemable convertible preferred stock	6,435
Net income	<u>\$ 24,192</u>

### *Accounting Change*

In April 1998, the American Institute of Certified Public Accountants issued Statement of Position ("SOP") 98-5, *Reporting on the Cost of Start-Up Activities*, which required that all organizational costs be expensed as incurred. The Company adopted this SOP effective January 1, 1999 and recorded a charge of \$364,000 as a cumulative effect of change in accounting principle.

### *Recently Issued Accounting Pronouncements*

In June 1998, the Financial Accounting Standards Board issued SFAS No. 133, *Accounting for Derivative Instruments and Hedging Activities*. The new standard, which was adopted on January 1, 2001, requires that all companies record derivatives on the balance sheet as assets or liabilities, measured at fair value. Gains or losses resulting from changes in the values of those derivatives are accounted for depending on the use of the derivative and whether it qualifies for hedge accounting. The adoption of this standard on January 1, 2001 had no impact on the Company's financial position or results of operations.

In June 2001, the Financial Accounting Standards Board issued SFAS No. 141, *Business Combinations*, and SFAS No. 142, *Goodwill and Other Intangible Assets*. SFAS No. 141 supersedes APB No. 16, *Business Combinations*, and SFAS No. 38, *Accounting for Preacquisition Contingencies of Purchased Enterprises* and requires that all business combinations be accounted for by a single method – the purchase method. SFAS No. 141 also provides guidance on the recognition of intangible assets identified in a business combination and requires enhanced financial statement disclosures. SFAS No. 142 adopts a more aggregate view of goodwill and bases the accounting for goodwill on the units of the combined entity into which an acquired entity is integrated. In addition, SFAS No. 142 concludes that goodwill and intangible assets that have indefinite useful lives will not be amortized but rather will be tested at least annually for impairment. Intangible assets that have finite lives will continue to be amortized over their useful lives. SFAS No. 141 is effective for all business combinations initiated after June 30, 2001. The adoption of SFAS No. 142 is required for fiscal years beginning after December 15, 2001 (the year 2002 for the Company), except for the nonamortization and amortization provisions which are required for goodwill and intangible assets acquired after June 30, 2001. The Company believes that the adoption of SFAS No. 141 and SFAS No. 142 will not have a material impact on the Company's financial position or results of operation.

In October 2001, the Financial Accounting Standards Board issued SFAS No. 144, "Accounting for the Impairment or Disposal of Long-Lived Assets." SFAS No. 144 supersedes previous guidelines for financial accounting and reporting for the impairment or disposal of long-lived assets and for segments of a business to be disposed of. The Company believes that the adoption of SFAS No. 144 will not have a material impact on the Company's financial position or results of operations.

### *Reclassifications*

Certain reclassifications have been made to prior year financial statements to conform to the 2001 presentation.

## **2. Marketable Securities**

The Company has classified its marketable securities as available-for-sale and, accordingly, carries such securities at aggregate fair value. At December 31, 2001 and 2000, all of the Company's marketable securities consisted of corporate debt securities. At December 31, 2001, all marketable securities had contractual maturities of one year or less.

At December 31, 2001, the aggregate fair value and amortized cost of the Company's marketable securities were \$444,000 and \$441,000, respectively. Gross unrealized gains and losses were \$3,000 and \$0, respectively, at December 31, 2001. At December 31, 2000, the aggregate fair value and amortized cost of the Company's

marketable securities were \$27.2 million. Gross unrealized gains and losses were \$11,000 and \$16,000, respectively, at December 31, 2000.

Gains and losses on investment security transactions are reported on the specific-identification method. Realized gains and losses on sales of marketable securities were not material during the years ended December 31, 2001, 2000 and 1999. Changes in market values resulted in an increase (decrease) in net unrealized gains (losses) of \$8,000, (\$5,000) and \$34,000 for the years ended December 31, 2001, 2000 and 1999, respectively.

### **3. Collaborative Research and Development Agreements**

In November 1995, the Company entered into an agreement with Hoechst Marion Roussel, S.A. ("HMR") (the "1995 Osteoporosis Agreement") to collaborate on the discovery and development of drugs to treat osteoporosis and related bone diseases, one of the Company's cell-signaling inhibitor programs. Under the 1995 Osteoporosis Agreement, the Company granted to HMR exclusive rights to develop and commercialize these drugs worldwide. Under the terms of this Agreement, HMR made an initial cash payment to the Company of \$10.0 million, agreed to provide research funding in equal quarterly amounts of \$1.0 million up to an aggregate of \$20.0 million over a five-year period and agreed to provide an aggregate of up to \$10.0 million upon the attainment of certain research milestones. This Agreement further provided for the payment of royalties to the Company based on product sales. Revenue recognized under the 1995 Osteoporosis Agreement amounted to \$6.0 million in 1999 including \$2.0 million for the achievement of the second research milestone.

Subsequently, in connection with the sale of the Company's 50% interest in the Genomics Center in 1999 (Note 4), all drug candidates and related technologies resulting from the 1995 Osteoporosis Agreement were assigned to the Company, and any further obligations of HMR to fund the Company's research were terminated.

### **4. Hoechst-ARIAD Genomics Center, LLC**

#### *Formation of the Genomics Center*

In March 1997, the Company entered into an agreement which established a 50%/50% joint venture with Aventis Pharmaceuticals, Inc. (formerly known as Hoechst Marion Roussel, Inc.) ("Aventis") to pursue functional genomics with the goal of identifying genes that encode novel therapeutic proteins and small-molecule drug targets (the "1997 Genomics Agreement"). The joint venture, named the Hoechst-ARIAD Genomics Center, LLC (the "Genomics Center"), was located at the Company's facility in Cambridge, Massachusetts. Under the terms of the 1997 Genomics Agreement, the Company and Aventis agreed to commit \$85.0 million to the establishment of the Genomics Center and its first five years of operation. The Company and Aventis agreed to jointly fund \$78.5 million of operating and related costs, and the Company agreed to invest up to \$6.5 million in leasehold improvements and equipment for use by the Company in conducting research on behalf of the Genomics Center. Through December 31, 1999, the Company had invested \$6.5 million in leasehold improvements and equipment and funded \$15.0 million in operating and related costs. Aventis committed to provide the Company with capital adequate to fund ARIAD's share of such costs through the purchase of up to \$49.0 million of the Company's series B convertible preferred stock over the five-year period, including an initial investment of \$24.0 million, which was completed in March 1997 and \$5.7 million which was completed in January 1999 (Note 7). Using a loan facility made available by Aventis, the Company borrowed \$1.8 million during 1999 to fund a portion of its investment obligations relating to the Genomics Center.

#### *Services Agreements*

The Company also entered into agreements with the Genomics Center to provide research and administrative services (the "Services Agreements") to the Genomics Center on a cost reimbursement basis. The Company's

costs of providing the research and administrative services to the Genomics Center were charged to research and development expense and general and administrative expense in the consolidated financial statements. Under the Services Agreements, the Company billed the Genomics Center for 100% of its cost of providing the research and administrative services; however, because the Company was providing 50% of the funding of the Genomics Center, the Company recognized as revenue only 50% of the billings to the Genomics Center. The remaining 50% was accounted for as a return of the Company's investment in the Genomics Center. Revenue recognized pursuant to the Services Agreements amounted to \$6.4 million and \$5.0 million for the years ended December 31, 1999 and 1998, respectively.

*Sale of the Company's 50% Interest in the Genomics Center*

On December 31, 1999, the Company completed the sale of its 50% interest in the Genomics Center to Aventis and received: (1) \$40.0 million in cash, (2) 3,004,436 shares of the Company's previously issued series B convertible preferred stock (which was immediately retired), (3) the forgiveness of \$1.9 million of long-term debt including accrued interest owed by the Company to Aventis, (4) drug candidates and related technologies resulting from the 1995 Osteoporosis Agreement (Note 3) and (5) the right to use certain genomics and bioinformatics technologies developed by the Genomics Center. In addition, the Company agreed to (1) sublease to Aventis approximately 35,000 square feet of laboratory and office space, for an amount equal to the Company's cost, for a period through July 2002, after which time the sublease rate shall be negotiated between the two parties for a period of up to five years, (2) assign equipment leases with aggregate rental payments of \$1.8 million to Aventis (Note 6), and (3) provide certain transitional laboratory support services.

The Company recorded a net gain on the sale of \$46.4 million recognizing proceeds of (1) \$40.0 million in cash, (2) \$8.5 million equivalent to the fair market value of the common stock underlying the series B convertible preferred stock, and (3) \$1.9 million of long-term debt and interest forgiven; offset by (1) \$2.3 million of unamortized leasehold improvements associated with laboratory space under sublease, and (2) \$1.6 million representing the Company's investment account and other costs of completing the sale.

The major components of the Genomics Center's results of operations for the year ending December 31, 1999 were as follows:

<i>In thousands</i>	
Revenues	\$ -
Operating expenses:	
ARIAD	12,936
Other	2,958
Net loss	<u>\$ (15,894)</u>
ARIAD's 50% share of net loss	\$ (7,947)
Elimination of intercompany transactions	<u>6,454</u>
ARIAD's equity in the net loss on the Genomics Center	<u>\$ (1,493)</u>

## 5. Long-Term Debt

Long-term debt was comprised of the following at December 31:

	<u>2001</u>	<u>2000</u>
	<i>In thousands</i>	
Bank term note at prime plus 1% (5.75%, at December 31, 2001) payable in monthly installments of \$100,000 plus interest, through January 1, 2005	\$ 7,500	\$ 4,900
General Electric Capital Corporation term note at 9.51% payable in monthly installments of \$25,104, which includes interest, through December 2004	790	
Less current portion	<u>(1,443)</u>	<u>(1,200)</u>
Long-term debt	<u>\$ 6,847</u>	<u>\$ 3,700</u>

The bank term note is collateralized by all assets of the Company with the exception of the assets to collateralize the General Electric Capital Corporation ("G.E.") term note. The Company may, at its option, pledge marketable securities under the bank term note, and, in such event, the interest rate would be adjusted to the equivalent of 90-day LIBOR plus 1.25%. No securities were pledged at December 31, 2001.

The bank term note agreement contains certain covenants that would require consent from the bank to change the Company's Chief Executive Officer, increase indebtedness, limit capital spending and stock redemption, prohibit dividend distributions, and require the Company to pledge its marketable securities or maintain minimum levels of tangible net worth of \$15.0 million, working capital of \$7.0 million and liquid assets of \$15.0 million plus the outstanding principal balance of the G.E. term note, all as defined.

The G.E. term note, which provides for borrowings up to \$1.2 million, is collateralized by certain assets of the Company. As of December 31, 2001, the Company has drawn down \$790,000 and has available \$410,000 remaining to be drawn down on the note. The G.E. term note contains a covenant that requires the Company to maintain a minimum unrestricted cash balance of \$10.0 million.

The annual aggregate future principal payments of the above debt agreements are \$1.4 million for 2002, \$1.5 million for 2003 and 2004 and \$3.9 million in 2005. Interest payments during 2001, 2000, and 1999 were \$255,000, \$204,000 and \$376,000 respectively.

## 6. Leases, Licensed Technology and Other Commitments

### *Facility Lease*

The Company conducts its operations in a 100,000 square foot office and laboratory facility under a ten-year non-cancelable operating lease. The lease expires in July 2002 and has two five-year options to extend. The Company has extended the lease for the first five-year extension period. The Company has sublet approximately 37,000 square feet of space to Aventis (Note 4) and another tenant. Rent expense, net of sublease income of \$950,000, \$1.2 million and \$264,000 for the years ended December 31, 2001, 2000, and 1999, respectively, amounted to \$654,000, \$477,000, and \$1.2 million, respectively. Future minimum annual rental payments, net of sublease income, for the next five years are approximately \$778,000 for 2002, \$710,000 for 2003 and \$747,000 for 2004, 2005 and 2006, respectively.

### *Equipment Leases*

The Company utilizes lease credit facilities from various equipment leasing companies to acquire equipment, which is resold to a lessor at cost, with no resulting gain or loss recognized. The lease agreements, which are classified as operating leases for financial reporting purposes, have terms ranging from three to five years with

various lease renewal or purchase options at the end of the initial term. The Company did not enter into any new equipment lease agreements in 2001 and 2000. During the year ended December 31, 1999, the Company entered into sales leaseback transactions amounting to \$309,000. Equipment rental expense for the years ended December 31, 2001, 2000 and 1999 amounted to \$897,000, \$933,000 and \$1.8 million, respectively. Some of the agreements contain covenants requiring the Company to maintain certain minimum levels of net worth, working capital and liquid assets. Minimum future rental payments under the initial terms of the leases are approximately \$741,000 for 2002, \$196,000 for 2003, and \$33,000 for 2004.

#### *Licensed Technology*

The Company and AGTI have entered into agreements with several universities under the terms of which the Company and AGTI have received exclusive licenses or options to technology and intellectual property. The agreements, which are generally cancelable by the Company and AGTI, provide for the payment of license fees and/or minimum payments, which are generally creditable against future royalties. Fees aggregated \$127,000, \$127,000 and \$105,000 for 2001, 2000 and 1999, respectively, and were charged to research and development expense and are expected to amount to approximately \$232,000 for 2002, and \$162,000 annually for 2003, 2004, 2005 and 2006. In addition, the agreements provide for payments upon the achievement of certain milestones in drug development. The agreements also require the Company to fund certain costs associated with the filing and prosecution of patent applications.

#### *Executive Compensation Plan*

The Company has an executive compensation plan which provides a deferred compensation benefit for certain executives and key employees. Under the plan, benefits are deferred and generally vest over four years. The benefits obligation is recorded as compensation and a liability based on the underlying fair market value of the obligation as it vests. The fair market value of the vested portion of this liability was \$474,000, \$299,000 and \$144,000 for December 31, 2001, 2000 and 1999, respectively.

#### *Other Commitments*

The Company has entered into various employment agreements with its senior officers. The agreements provide for aggregate annual base salaries of \$2.2 million and remaining terms of employment of up to two years.

## **7. Stockholders' Equity**

#### *Preferred Stock*

The Company has authorized 10.0 million shares of preferred stock which the Board of Directors is empowered to designate and issue in different series. At December 31, 2001, the Board of Directors had designated 500,000 shares as series A preferred stock, and 9.5 million shares remained undesignated.

#### *Series B Convertible Preferred Stock ("Series B Preferred Stock")*

In connection with the 1997 Genomics Agreement, on March 18, 1997, Aventis purchased 2,526,316 shares of the Company's Series B Preferred Stock for \$24.0 million, and on January 5, 1999, Aventis purchased an additional 478,120 shares of Series B Preferred Stock for \$5.7 million. In connection with the sale of the Company's interest in the Genomics Center, all shares of Series B Preferred Stock were redeemed by the Company and retired.

#### *Series C Redeemable Convertible Preferred Stock ("Series C Preferred Stock")*

On November 9, 1998, the Company issued 5,000 shares of the Company's Series C Preferred Stock to two institutional investors (the "Investors") and received proceeds of approximately \$5 million. Each share of



Series C Preferred Stock had a liquidation value of \$1,000, plus an additional amount equal to a 5% per annum accretion amount, accrued from the date of issue, and was convertible into common stock of the Company, at a conversion price equal to the lower of a variable conversion price or \$2.09 per share.

On December 31, 1999, the Company repurchased 2,000 shares of Series C Preferred Stock from one of the Investors for an aggregate cash payment of \$3.4 million. On January 14, 2000, the Company completed the repurchase of the remaining 3,000 shares of its Series C Preferred Stock for an aggregate consideration of \$6.9 million plus 1,078,038 shares of common stock. The aggregate premium of \$6.2 million paid on both transactions has been included in the 1999 consolidated statements of operations as repurchase and accretion costs attributable to redeemable convertible preferred stock. Redeemable convertible preferred stock was carried at redemption cost at December 31, 1999.

#### *Common Stock – Sales*

In 2001, the Company sold an aggregate of approximately 2.6 million shares of common stock and received net proceeds of \$13.8 million in addition to the sale of registered shares mentioned below. In 2000, the Company sold an aggregate of 857,000 shares of common stock and received net proceeds of \$9.7 million. All of the approximately 3.5 million shares sold were publicly registered.

#### *Common Stock – Shelf Registration*

On June 22, 2001, the Company filed a shelf registration statement with the Securities and Exchange Commission (the “SEC”) for the issuance of up to 4.5 million registered shares of its common stock, which was declared effective by the SEC on August 1, 2001. The registered shares are available for sale at the Company’s discretion. On October 31, 2001, the Company sold 1,927,712 registered shares of its common stock to institutional investors at a price of \$4.15 per share and received gross proceeds of \$8.0 million before commissions and expenses of \$464,000. At December 31, 2001 the Company had 2,572,287 registered shares remaining available for sale.

#### *Redemption of Warrants*

The Company received funds aggregating approximately \$11.6 million from the exercise of approximately 1.4 million of its publicly traded warrants during the first and second quarters of 2000. Each warrant was exercisable for one share of common stock at an exercise price of \$8.40 per share. The warrants had been called for redemption effective April 27, 2000. At December 31, 2001 and 2000, there were no warrants outstanding.

#### *Stockholder Rights Plan*

The Board of Directors of the Company adopted a new Rights Agreement, dated as of June 8, 2000 (the “2000 Rights Agreement”), between the Company and State Street Bank and Trust Company, as Rights Agent, and approved the declaration of a dividend distribution of one Preferred Share Purchase Right (a “Right”) on each outstanding share of its Common Stock. In general, the Rights become exercisable if a person or group hereafter acquires 15% or more of the Common Stock of the Company or announces a tender offer for 15% or more of the Common Stock. The Board of Directors will, in general, be entitled to redeem the Rights at one cent per Right at any time before any such person hereafter acquires 15% or more of the outstanding Common Stock. The plan is designed to protect the Company’s stockholders in the event that an attempt is made to acquire the Company without an offer of fair value.

If a person hereafter acquires 15% or more of the outstanding Common Stock of the Company (the “Acquiring Person”), each Right will entitle its holder to purchase, for an initial exercise price of \$65, a number of shares of Common Stock having a market value at that time of twice the Right’s exercise price. Rights held by the Acquiring Person will become void. If the Company is acquired in a merger or other business combination

transaction after a person acquires 15% or more of the Company's Common Stock, each Right will entitle its holder to purchase, at the Right's then-current exercise price, a number of the acquiring company's common shares having a market value at that time of twice the Right's exercise price.

The dividend distribution of Rights was payable on July 19, 2000 to shareholders of record on June 19, 2000. The Rights will expire in ten years. The Rights distribution is not taxable to the Company's stockholders.

The Board of Directors also adopted two amendments to the Rights Agreement dated December 15, 1994, (the "1994 Rights Agreement"), between the Company and State Street Bank and Trust Company, as Rights Agent. As a result of these amendments, the adoption of the 2000 Rights Agreement and the setting of a record date to distribute new Rights, the 1994 Rights Agreement is no longer in effect.

#### *Minority Interest in Subsidiary*

The 20% minority interest in AGTI includes shares owned by Stanford University and Harvard University (which together own 2%) issued in 1995 in connection with a license agreement and shares issued to option holders (18%), including members of the Company's management, Board of Directors, and certain consultants. Additional stock options are outstanding and, if exercised, would increase the minority interest to 21% (Note 8).

### **8. Stock Option and Stock Purchase Plans**

#### *ARIAD Stock Option and Stock Plans*

The Company's 1991, 1994 and 2001 Stock Option and Stock Plans (the "Plans") provide for the granting of nonqualified and incentive stock options to purchase up to a maximum of 7,615,714 shares of common stock to officers, directors, employees and consultants of the Company. Options become exercisable as specified in the related option agreement, typically over a four-year period, and expire ten years from the date of grant.

Transactions under the Plans for the years ended December 31, 1999, 2000 and 2001 are as follows:

	Number of Shares	Weighted Average Exercise Price Per Share
Options outstanding, January 1, 1999	4,538,737	\$3.34
Granted	2,128,095	1.03
Forfeited	(1,555,588 )	3.09
Exercised	(41,875 )	1.59
Options outstanding, December 31, 1999	5,069,369	2.48
Granted	553,300	10.99
Forfeited	(226,668 )	4.04
Exercised	(1,915,641 )	2.40
Options outstanding, December 31, 2000	3,480,360	3.77
Granted	1,554,220	5.61
Forfeited	(120,688 )	5.75
Exercised	(274,110 )	1.51
Options outstanding, December 31, 2001	4,639,782	\$4.47
Options exercisable, December 31, 1999	3,536,268	\$2.47
December 31, 2000	2,268,950	\$2.64
December 31, 2001	2,650,830	\$3.31

The following table sets forth information regarding options outstanding at December 31, 2001:

Range of Exercise Prices	Number of Shares	Weighted Average Exercise Price	Weighted Average Remaining Life (years)	Number of Option Shares Currently Exercisable	Weighted Average Exercise Price for Currently Exercisable
\$ .75 - 1.25	552,239	\$ .77	7.8	530,639	\$ .76
1.34 - 2.31	1,196,575	1.76	4.5	941,287	1.84
2.68 - 4.88	1,005,066	4.30	6.5	705,937	4.21
4.89 - 8.00	1,544,902	6.05	9.0	376,469	6.33
12.56 - 14.63	341,000	13.41	8.5	96,498	13.31
\$ .75 - 14.63	4,639,782	\$ 4.47	7.1	2,650,830	\$ 3.31

As described in Note 1, the Company uses the intrinsic value method to measure compensation expense associated with grants of stock options to employees. The Company has also issued options to consultants, which are included in the table above. The unearned portion of these awards is classified as a component of stockholder equity and is listed as “deferred compensation” on the consolidated balance sheet. On a proforma basis, had the Company used the fair value method to measure compensation, the net income (loss) and net income (loss) per share would have been reported as follows:

<i>In thousands (except per share data)</i>	<b>Years ended December 31,</b>		
	<b>2001</b>	<b>2000</b>	<b>1999</b>
Basic:			
Proforma net income (loss) attributable to common stockholders	\$ (23,376)	\$ (16,667)	\$ 16,311
Proforma net income (loss) per share	\$ (.80)	\$ (.64)	\$ .74
Diluted:			
Proforma net income (loss) attributable to common stockholders plus repurchase and accretion costs attributable to redeemable convertible preferred stock	\$ (23,376)	\$ (16,667)	\$ 22,746
Proforma net income (loss) per share	\$ (.80)	\$ (.64)	\$ .66

At December 31, 2001, the Company has 449,759 options available to be issued at future dates under the Plans.

The above disclosure, required by SFAS No. 123, includes only the effect of grants made subsequent to January 1, 1996. For purposes of calculating the above disclosure, the fair value of options on their grant date was measured using the Black-Scholes option pricing model. Key assumptions used to apply this pricing model included a risk-free interest rate of 4.2% for 2001, 5.9% for 2000 and 5.5% for 1999, expected lives of the option grants ranging from one to six years and expected rates of volatility for the underlying stock of 108% for 2001, 109% for 2000 and 100% for 1999. Using this model, the weighted average fair value per option for all options granted to employees in 2001, 2000 and 1999 was \$4.71, \$9.35 and \$1.09, respectively.

#### *ARIAD Gene Therapeutics, Inc. Stock Option Plan*

The Company’s subsidiary, AGTI, adopted a stock option plan in 1993 substantially similar to the Plans and reserved 1,785,714 shares of AGTI’s common stock for issuance pursuant to such plan. At December 31, 2001, options with respect to 87,428 shares of AGTI’s common stock (all granted in 1994) were outstanding at an exercise price of \$.42 per share, and all option shares were exercisable. During 2001, no options were exercised or forfeited. During 2000, 758,282 options were exercised with aggregate proceeds of \$318,000, and 25,000 option shares were forfeited. During 1999, 89,285 options were exercised with aggregate proceeds of \$37,000, and 207,142 option shares were forfeited. As of December 31, 2001, AGTI had 5,195,779 shares of its common stock outstanding.

#### *Employee Stock Purchase Plan*

In 1997, the Company adopted the 1997 Employee Stock Purchase Plan and reserved 500,000 shares of common stock for issuance under this plan. Under this plan, substantially all of its employees may, through payroll withholdings, purchase shares of the Company’s stock at a price of 85% of the lesser of the fair market value at the beginning or end of each three-month withholding period. In 2001, 2000 and 1999, 28,985, 20,049, and 51,259 shares of common stock were issued under the plan, respectively.

## 9. Income Taxes

At December 31, 2001, the Company had available for federal tax reporting purposes, net operating loss carryforwards of approximately \$110.1 million, which expire commencing in 2009 and for state tax reporting purposes, net operating loss carryforwards of approximately \$85.2 million, which expire commencing in 2002. The Company also had federal research and development credit carryovers of approximately \$6.0 million, which expire commencing in 2006. Both the net operating loss carryforwards and credits are subject to certain limitations under federal tax law.

The components of deferred income taxes were as follows at December 31:

<i>In thousands</i>	<u>2001</u>	<u>2000</u>
Deferred tax liabilities:		
Intangible and other assets	\$ 2,091	\$ 1,882
Organizational costs		2
Total deferred tax liabilities	<u>2,091</u>	<u>1,884</u>
Deferred tax assets:		
Net operating loss carryforwards	42,590	34,707
Federal and State tax credit carryovers	10,808	9,860
Depreciation	3,612	3,416
Other	408	255
Total deferred tax assets	<u>57,418</u>	<u>48,238</u>
Deferred tax assets, net	55,327	46,354
Valuation allowance	(55,327)	(46,354)
Total deferred taxes	<u>\$ 0</u>	<u>\$ 0</u>

Although the Company earned taxable income in 1999 due to the gain on sale of the Genomics Center, it was able to utilize net operating loss carryforwards to eliminate substantially all taxes due. Since the Company has not yet achieved sustained profitable operations, management believes the tax benefits as of December 31, 2001 and 2000 do not satisfy the realization criteria set forth in SFAS No. 109 and has recorded a valuation allowance for the entire net deferred tax asset. The increase in the valuation allowance of \$8.9 million in 2001 and \$11.6 million in 2000 resulted primarily from net operating loss carryforwards and tax credit carryovers that resulted from operations in those years. The decrease in the valuation allowance of \$8.4 million in 1999 resulted from the utilization of net operating loss carryforwards.

## 10. Litigation

The Company was named as a defendant in a purported class action lawsuit commenced in June 1995 in the U.S. District Court for the Southern District of New York. The action named as defendants the Company; the underwriter of the Company's initial public offering and a market maker in the Company's stock, D. Blech & Co.; the managing director and sole shareholder of D. Blech & Co. and a former director of the Company, David Blech; certain other directors of the Company, and the qualified independent underwriter for the initial public offering, Shoenberg Hieber, Inc. ("SHI").

Counsel for the plaintiff class, counsel for the Company and the named director defendants, excluding David Blech, (the "Company Defendants") and counsel for SHI have executed a stipulation of settlement in the action (the "Proposed Settlement"). The Proposed Settlement, in substance, contemplates a payment of \$620,000 as consideration for plaintiffs' consent to entry of judgment dismissing the action with prejudice and barring "contribution-type" claims against the Company Defendants by non-settling parties. The

Proposed Settlement further is subject to the Court's approval of that stipulation as fair, adequate and reasonable, and to entry of an appropriate judgment of dismissal in the action and in a related action entitled In re: Blech Securities Litigation, 94 Civ. 7696 (RWS), from which the Court previously ordered the Company dismissed as a defendant. The amount the Company has contributed to a legal escrow account was not material, and accordingly, no liability is recorded on the 2001 balance sheet.

On May 19, 1999, the Company filed suit in the Massachusetts Superior Court against Michael Z. Gilman, Ph.D. ("Dr. Gilman"), the Company's former Chief Scientific Officer, seeking equitable relief for breach of his employment agreements in accepting a position as the research director of molecular biology at Biogen, Inc. ("Biogen"). The Superior Court issued a temporary injunction on May 19, 1999 restraining Dr. Gilman from using any of the Company's confidential information in his new employment. On June 21, 1999, Dr. Gilman filed counterclaims against the Company seeking an order awarding damages for breach of contract and barring the Company from enforcing any provisions of its employment agreements with Dr. Gilman. On May 26, 1999 Biogen filed a motion to intervene as a defendant in the action which the Superior Court granted on August 2, 1999. Discovery in the case has been completed, and Summary Judgment Motions have been filed, heard and ruled upon. The Company's claim against Dr. Gilman and Dr. Gilman's counterclaims will now proceed to trial. No trial date has been set. The ultimate outcome of the litigation with Dr. Gilman is not determinable or estimatable at this time, and accordingly, no liability is recorded on the balance sheets.

**ITEM 9: CHANGES IN AND DISAGREEMENTS WITH ACCOUNTANTS ON ACCOUNTING AND FINANCIAL DISCLOSURE**

Not Applicable.

## PART III

### ITEM 10: DIRECTORS AND EXECUTIVE OFFICERS OF THE REGISTRANT

The directors, officers and key employees of the Company are as follows:

<u>Name</u>	<u>Age</u>	<u>Position</u>
Harvey J. Berger, M.D.....	51	Chairman of the Board of Directors, Chief Executive Officer and President
Sandford D. Smith .....	54	Vice Chairman of the Board of Directors
Laurie A. Allen, Esq.. .....	41	Senior Vice President, Chief Legal Officer, and Secretary
David L. Bernstein, Esq.. .....	49	Senior Vice President and Chief Patent Counsel
Fritz Casselman.....	52	Senior Vice President and Chief Business Officer
John D. Iuliucci, Ph.D.....	59	Senior Vice President, Drug Development
Timothy P. Clackson, Ph.D.....	36	Vice President, Gene Therapy and Genomics
David C. Dalgarno, Ph.D. ....	44	Vice President, Physical and Chemical Sciences
Maryann G. Krane .....	42	Vice President, Regulatory Affairs
Tomi K. Sawyer, Ph.D.....	47	Vice President, Drug Discovery
Thomas A. Pearson.....	60	Senior Advisor, Corporate Communications and Planning
Brian A. Lajoie.....	55	Interim Chief Financial Officer
Joseph Bratica .....	38	Director, Finance and Controller
Vaughn D. Bryson .....	63	Director
John M. Deutch, Ph.D. ....	63	Director
Jay R. LaMarche .....	55	Director
Ralph Snyderman, M.D. ....	62	Director
Raymond S. Toubh .....	75	Director

*Harvey J. Berger, M.D.* is our principal founder and has served as our Chairman of the Board, Chief Executive Officer and President since April 1991. From 1986 to 1991, Dr. Berger held a series of senior management positions at Centocor, Inc., a biotechnology company, including Executive Vice President and President, Research and Development Division. He also has held senior academic and administrative appointments at Emory University, Yale University and the University of Pennsylvania and was an Established Investigator of the American Heart Association. Dr. Berger is a director of PTC Therapeutics, Inc., a closely held biotechnology company. Dr. Berger received his A.B. degree in Biology from Colgate University and his M.D. degree from Yale University School of Medicine and did further medical and research training at the Massachusetts General Hospital and Yale-New Haven Hospital.

*Sandford D. Smith*, one of our Directors since October 1991 and our Vice Chairman since January 1999, is Corporate Vice President and President, Genzyme Europe and International, Genzyme Corporation. From October 1997 to December 2000, he was President, Therapeutics International, Genzyme Corporation, and from May 1996 to September 1996, Vice President and General Manager, Specialty Therapeutics and International Group, Genzyme Corporation, a biotechnology company. Mr. Smith was President and Chief Executive Officer and a Director of Repligen Corporation, a biotechnology company, from 1986 to March 1996. Mr. Smith previously held a number of positions with Bristol-Myers Squibb and Company from 1977 to 1986, including, most recently, Vice President of Corporate Development and Planning for the United States Pharmaceutical and Nutritional Group. Mr. Smith is a Director of CSPI, a software company. Mr. Smith earned his B.A. degree from the University of Denver.

*Laurie A. Allen, Esq.* has served as our Senior Vice President and Chief Legal Officer since March 2002 and has served continuously as our Secretary since January 1999. Previously, from January 1999 to December 1999, she served as our Senior Vice President, Corporate Development and Legal Affairs and General Counsel. From January 2000 to February 2002, Ms. Allen was Senior Vice President, Business Development and Legal Affairs at Alexandria Real Estate Equities, Inc., a real estate investment trust. Previously, she was a partner with Brobeck, Phleger and Harrison, LLP, a leading technology law firm, from January 1996 to December 1998. She also was an associate with Brobeck, Phleger & Harrison, LLP from February 1991 to December 1995. Ms. Allen received her A.B. degree in History from the University of California, Los Angeles, her L.L.M. degree in taxation from New York University and her J.D. degree from Emory University School of Law.

*David L. Berstein, Esq.* has served as our Senior Vice President and Chief Patent Counsel since June 2000. Previously, he served as our Vice President and Chief Patent Counsel from September 1993 to June 2000. Prior to joining us, from 1990 through 1993, Mr. Berstein was Patent Counsel at BASF Bioresearch Corporation, a biotechnology company, where he was responsible for intellectual property matters, including patents and licensing. From 1985 to 1990, Mr. Berstein was a patent attorney at Genetics Institute, Inc., a biotechnology company, where he was involved in various aspects of the patent process from patent procurement through litigation. Mr. Berstein joined Genetics Institute from the law firm of Cooper & Dunham. Mr. Berstein received his B.S. degree from the University of Michigan and his J.D. degree from Fordham University School of Law.

*Fritz Casselman* has served as our Senior Vice President and Chief Business Officer since February 2001. From February 2000 to January 2001, Mr. Casselman was Senior Vice President, Strategy and Corporate Development at Avant Immunotherapeutics Inc., a biotechnology company. From 1997 to 2001, Mr. Casselman was Director of Worldwide Business Development at SmithKline Beecham, plc, a pharmaceutical company; from 1988 to 1996, Vice President and consultant to Cambridge Biotech Corporation, a biotechnology company; and from 1982 to 1988 an associate and then a partner at the law firm of Bromberg, Sunstein and Casselman. Mr. Casselman received his B.A. degree from the University of Wisconsin (Madison) and his J.D. degree from Boston University School of Law.

*John D. Iuliucci, Ph.D.* has served as our Senior Vice President, Drug Development since January 1999. Previously, he also served as our Vice President, Drug Development from October 1996 to December 1998 and our Vice President, Preclinical Development from June 1992 to September 1996. Prior to joining us, Dr. Iuliucci was Director of Preclinical Pharmacology and Toxicology at Centocor, Inc., a biotechnology company, from 1984 to 1992. From 1975 to 1984, Dr. Iuliucci headed the Drug Safety Evaluation Department at Adria Laboratories, a pharmaceutical company. He was a Senior Toxicologist at the Warner-Lambert Pharmaceutical Research Institute from 1972 to 1975. Dr. Iuliucci received his B.S. degree in Pharmacy and M.S. and Ph.D. degrees in Pharmacology from Temple University.

*Timothy P. Clackson, Ph.D.* has served as our Vice President, Gene Therapy and Genomics since June 2000. Previously he served as our Director, Gene Therapy from August 1999 to June 2000 and as our Department Head, Gene Therapy Biology from March 1999 to August 1999. Prior to joining us in December 1994, Dr. Clackson was a postdoctoral fellow at Genentech, Inc., a biotechnology company, from 1991 to 1994, where he studied the molecular basis for human growth hormone function. Dr. Clackson received his B.A. degree in Biochemistry from the University of Oxford. Dr. Clackson received his Ph.D. in Biology from the University of Cambridge, for research conducted at the MRC Laboratory of Molecular Biology into antibody engineering and the development of phage display technology.

*David C. Dalgarno, Ph.D.* has served as our Vice President, Physical and Chemical Sciences since November 1999. Previously, he served as our Director, Physical and Chemical Sciences from September 1998 to November 1999 and as our Director, Spectroscopy from October 1996 to August 1998. Prior to joining us in March 1992, Dr. Dalgarno was a scientist at Schering-Plough Corp. focusing on protein structure determination by nuclear magnetic resonance. Dr. Dalgarno received his B.A. and Ph.D. degrees in



Chemistry from the University of Oxford. He received his postdoctoral training in Molecular Biophysics and Biochemistry at Yale University.

*Maryann G. Krane* has served as our Vice President, Regulatory Affairs since May 2001. From September 2000 to May 2001, she served as Senior Director, Regulatory Affairs and Quality Assurance at Avant Immunotherapeutics, Inc., a biotechnology company. From 1986 to 1992 and from 1993 to 2000, Ms. Krane held various positions in regulatory affairs and research at Genetics Institute, Inc., currently a unit of American Home Products Corporation, a diversified healthcare company. Most recently, she was Head, Regulatory Affairs, Global Development of Hemophilia and Oncology Products at Genetics Institute. From August 1992 to April 1993, she was Manager, Regulatory Affairs at Genzyme Corporation, a biotechnology company. Ms. Krane received her B.S. degree in Microbiology from the University of Massachusetts at Amherst, MA.

*Tomi K. Sawyer, Ph.D.* has served as our Vice President, Drug Discovery since January 1999. Previously, he served as our Director, Drug Discovery – Signal Transduction from October 1997 to December 1998. From July 1993 to September 1997, he was Head and Associate Research Fellow, Structure-Based Design and Chemistry at Parke-Davis Pharmaceutical Research a Division of Warner-Lambert Company, a pharmaceutical company, and Section Director, Peptide and Peptidomimetic Chemistry at Parke-Davis from July 1991 to July 1993. Dr. Sawyer received his B.S. degree in Chemistry from Moorhead State University and his Ph.D. degree in Organic Chemistry from the University of Arizona.

*Thomas A. Pearson* has served as our Senior Advisor, Corporate Communications and Planning since January 2001. Previously, he served as our corporate communications consultant since 1992. Mr. Pearson was an independent business consultant since 1983, specializing in biotechnology and high-technology companies. Previously, Mr. Pearson held various management positions in the television stations division of CBS, an entertainment and broadcasting company. Mr. Pearson received his B.A. degree in liberal arts from Wheaton College.

*Brian A. Lajoie* has served as our Interim Chief Financial Officer since October 2001 and also served in this position from October 2000 to February 2001. Mr. Lajoie has been a financial management consultant for more than two years. From August 1989 to October 1999, he was Vice President, Finance at Biopure Corporation, a biopharmaceutical company. Mr. Lajoie received his B.A. degree in Economics from the University of Massachusetts at Amherst, MA.

*Joseph Bratica* has served as our Director of Finance and Controller since January 1999. Previously, he served as our Assistant Controller from January 1997 to December 1998 and as our Accounting Manager from August 1994 to December 1996. Prior to joining us, he was Accounting Manager at Creative BioMolecules, Inc., a biotechnology company, from 1992 to 1994. Mr. Bratica received his B.A. degree in Accounting from Suffolk University.

*Vaughn D. Bryson*, one of our Directors since February 1995, is President of Life Science Advisors, Inc., a healthcare consulting company. Mr. Bryson was a thirty-two year employee of Eli Lilly & Co., a pharmaceutical company, from 1961 to 1993 and served as President and Chief Executive Officer of Eli Lilly from 1991 to 1993. He served as Executive Vice President of Eli Lilly from 1986 until 1991. He also served as a member of Eli Lilly's Board of Directors from 1984 until his retirement in 1993. Mr. Bryson was Vice Chairman of Vector Securities International Inc., an investment-banking firm, from April 1994 to December 1996. He also is a Director of Chiron Corporation, a biotechnology company, AtheroGenics, Inc., a biotechnology company, Amylin Pharmaceuticals, Inc., a biotechnology company, and Quintiles Transnational Corporation, a pharmaceutical services company. He received his B.S. degree in Pharmacy from the University of North Carolina and completed the Sloan Program at the Stanford University Graduate School of Business.

*John M. Deutch, Ph.D.*, one of our Directors since March 1997, is an Institute Professor at the Massachusetts Institute of Technology. From 1992 to 1997, he previously served as Director of Central Intelligence, Deputy Secretary of Defense, and Undersecretary of Defense (Acquisition and Technology). Prior to this, he was Provost of the Massachusetts Institute of Technology, Dean of the School of Science, Chairman of the Department of Chemistry and the Karl Taylor Compton Professor of Chemistry. Mr. Deutch is a Director of Citicorp, a financial services company, CMS Energy Corporation, an energy company, Cummins Engine Company, Inc., a manufacturer of engines and engine components, Raytheon, Inc., a defense and commercial electronics company, and Schlumberger Ltd., an oil and gas equipment services company. Mr. Deutch received his B.A. degree from Amherst College and his D.Sc. degree from the Massachusetts Institute of Technology and was a postdoctoral fellow at the National Institutes of Health.

*Jay R. LaMarche*, one of our Directors since January 1992, has served as a financial advisor since November 2000. Previously, he served as our Chief Financial Officer and Treasurer from January 1992 to November 2000 and as our Executive Vice President from March 1997 to November 2000. Mr. LaMarche was our Senior Vice President, Finance from January 1992 to February 1997. Prior to joining us, he was Chief Financial Officer and a Director of ChemDesign Corporation, a fine chemicals manufacturer. Previously, Mr. LaMarche was a partner with Deloitte Haskins & Sells, a public accounting firm. Mr. LaMarche received his B.B.A. degree in Public Accountancy from the University of Notre Dame and served as an officer in the United States Navy.

*Ralph Snyderman, M.D.*, one of our Directors since June 1998, has been Chancellor for Health Affairs and Dean, School of Medicine at Duke University since March 1989, and President and Chief Executive Officer of Duke University Health System since July 1998. He was formerly Senior Vice President of Medical Research and Development at Genentech, Inc., a biotechnology company, from January 1987 to May 1989. Dr. Snyderman is a Director of Proctor and Gamble, Inc., a consumer products and healthcare company. Dr. Snyderman received his B.S. degree from Washington College, Chestertown, Maryland and his M.D. degree from the State University of New York.

*Raymond S. Troubh*, one of our Directors since October 1991, has been a financial consultant for more than five years. Prior to this, he was a general partner of Lazard Freres & Co., an investment banking firm, and a governor of the American Stock Exchange. Mr. Troubh is a Director of Diamond Offshore Drilling, Inc., a contract drilling company, Enron Corporation, an energy trading company, General American Investors Company, Inc., an investment trust company, Gentiva Health Services, Inc., a healthcare provider, Health Net, Inc., a managed healthcare company, Petrie Stores Corporation, a liquidating trust, Starwood Hotels & Resorts, Inc., a hotel operating company, Hercules Incorporated, a specialty chemicals company, Triarc Companies, Inc., a holding company, and WHX Corporation, a steel products company. He received his A.B. degree from Bowdoin College and his L.L.B. degree from Yale Law School.

## **Section 16(a) Beneficial Ownership Reporting Compliance**

Section 16(a) of the Exchange Act requires our directors, executive officers and beneficial owners of more than 10% of our Common Stock to file reports of ownership and changes of ownership with the Commission on Forms 3, 4 and 5. We believe that during the fiscal year ended December 31, 2001, our directors, executive officers and beneficial owners of more than 10% of our Common Stock complied with all applicable filing requirements. In making these disclosures, we have relied solely on copies of such reports furnished to us.

## **ITEM 11: EXECUTIVE COMPENSATION**

The following table sets forth aggregate amounts of compensation paid or accrued by us for the years ended December 31, 2001, 2000 and 1999 for services rendered in all capacities, by our Chief Executive Officer and our four next most highly compensated executive officers (as of December 31, 2001).

### Summary Compensation Table

Name and Principal Position	Year	Annual Compensation		Long-Term Compensation	All Other Compensation (2)
		Base Salary	Other Compensation (1)	Number of Shares Underlying Options	
Harvey J. Berger, M.D. <i>Chairman, Chief Executive Officer and President</i>	2001	\$ 395,000	\$ -0-	115,000	\$ 3,318
	2000	363,000	-0-	100,000	3,400
	1999	330,000	-0-	250,000	3,200
David L. Bernstein, Esq. <i>Senior Vice President and Chief Patent Counsel</i>	2001	221,000	60,000	60,000	3,951
	2000	200,000	47,000	35,000	3,082
	1999	190,000	25,000	67,000	2,889
Fritz Casselman <i>Senior Vice President and Chief Business Officer (3)</i>	2001	180,923	-0-	200,000	3,896
	2000	-0-	-0-	-0-	-0-
	1999	-0-	-0-	-0-	-0-
John D. Iulucci, Ph.D. <i>Senior Vice President, Drug Development</i>	2001	221,000	60,000	55,000	5,016
	2000	207,500	37,000	-0-	16,485
	1999	200,000	30,000	90,000	4,281
Lee C. Steele <i>Senior Vice President (3)</i>	2001	183,346	-0-	-0-	3,858
	2000	-0-	-0-	27,500	-0-
	1999	-0-	-0-	-0-	-0-

- (1) The amounts listed are for deferred compensation awarded under our 1997 Executive Compensation Plan, a non-qualified, unfunded, deferred compensation plan. The amounts awarded vest over a four-year period commencing on the first anniversary of the date of the award.
- (2) The amounts listed for each year consist of our matching contributions of up to \$3,400 per year under our 401(k) Plan and, in the case of Mr. Bernstein, Mr. Casselman, Dr. Iulucci and Mr. Steele, include the aggregate difference between the fair market value and the purchase cost of common stock purchased during fiscal year 2001 under our 1997 Employee Stock Purchase Plan. Dr. Berger is not eligible to participate in our Employee Stock Purchase Plan.
- (3) Mr. Casselman's employment was effective February 6, 2001. Mr. Steele's employment was effective February 1, 2001, and he resigned as senior vice president on March 18, 2002.

### Stock Options

The following table sets forth information regarding each stock option granted during the fiscal year ended December 31, 2001 to each of the named executive officers.

### Stock Option Grants in Last Fiscal Year

Name	Individual Grants				Potential Realizable Value at Assumed Annual Rates of Stock Price Appreciation for Option Term (2)	
	Number of Shares Underlying Options Granted (1)	Percent of Total Options Granted to Employees in Fiscal Year	Exercise Price (per share)	Expiration Date	5%	10%
Harvey J. Berger, M.D.	115,000 (3)(4)	7.4%	\$ 4.80	07/26/11	\$347,150	\$879,746
David L. Bernstein, Esq.	60,000 (3)(4)	3.9%	5.65	06/07/11	240,805	540,279
Fritz Casselman	200,000 (3)(4)	12.9%	7.38	01/24/11	1,047,751	2,350,770
John D. Iulucci, Ph.D.	55,000 (3)(4)	3.5%	5.65	06/07/11	220,738	495,255
Lee C. Steele	-0-					

- (1) Options to purchase shares of our common stock.
- (2) These amounts, based on assumed appreciation rates of 5% and 10% as prescribed by the rules of the SEC, are for illustration purposes only and are not intended to forecast possible future appreciation, if any, of our stock price.
- (3) Vest annually over four years commencing on the first anniversary of the award.
- (4) Dr. Berger's and Mr. Bernstein's options were granted under the 2001 Stock Plan. Mr. Casselman's and Dr. Iulucci's options were granted under the 1991 Stock Option Plan for Employees.

## Aggregated Option Exercises in Last Fiscal Year and Fiscal Year-End Option Values

The following table provides information regarding the exercise of options by each of the named executive officers during the fiscal year ended December 31, 2001. In addition, this table includes the number of shares covered by both exercisable and unexercisable stock options as of December 31, 2001 and the values of "in-the-money" options, which values represent the positive spread between the exercise price of any such option and either the actual or estimated fair market value of the underlying security, as applicable.

### Aggregated Option Exercises in Last Fiscal Year and Fiscal Year-End Option Values

Name	Shares Acquired on Exercise (#)	Value Realized	No. of Shares Underlying Unexercised Options at Fiscal Year-End Exercisable/ Unexercisable	Value of Unexercised In-the-Money Options at Fiscal Year-End
Harvey J. Berger, M.D.	0	\$0	589,714/250,000 (1) (2)	\$1,884,138/235,750 (4)
	0	0	1,402/0 (3)	0/0 (5)
David L. Berstein, Esq.	0	0	122,964/102,750 (1)	440,491/51,823 (4)
	0	0	112/0 (3)	0/0 (5)
Fritz Casselman	0	0	0/200,000 (1)	0/0 (5)
John D. Iuliucci, Ph.D.	0	0	209,178/78,750 (1)	731,354/69,821 (4)
	0	0	280/0 (3)	0/0 (5)
Lee C. Steele	0	0	27,500/0 (1)	0/0 (4)

(1) Options to purchase shares of our common stock.

(2) Includes options to purchase 235,714 shares of our common stock held by The Berger Family Trust, all of which are exercisable.

(3) Options to purchase common stock of our subsidiary, AGTI.

(4) Based upon a fair market value of \$5.33 per share of common stock, which was the closing price of a share of our common stock on the NASDAQ National Market on December 31, 2001.

(5) Based upon an estimated value of the common stock of AGTI, for which there was no public market on December 31, 2001.

## Director Compensation

Effective as of 2001, non-employee directors receive an annual award of options to purchase 15,000 shares of our common stock, which are exercisable on the date of grant. On December 3, 2001, each non-employee director was awarded options to purchase 15,000 shares of our common stock at \$4.09 per share, pursuant to our stock options plans. Such options were exercisable on the grant date. On July 1, 2001, each non-employee director was also awarded a one-time grant of 8,500 options for each year of service on the Board of Directors, up to a maximum of three years of service and a total of 25,500 options to purchase shares of our common stock at \$5.00 per share. Such options were exercisable on the date of grant. Mr. Smith receives \$4,000 per month for his services as Vice Chairman of the Board of Directors. No other non-employee director receives any cash compensation for service on the Board of Directors or its committees. Directors are reimbursed for their expenses for each meeting they attend.

## Executive Employment Agreements

Dr. Berger, our Chairman of the Board of Directors, Chief Executive Officer and President, has an employment agreement with us which commenced in January 1992 and terminates in December 2004. Dr. Berger's employment agreement is automatically renewable for successive three-year terms unless terminated by either party. The agreement provides that he shall be employed as our Chief Executive Officer and President, shall be nominated for election to our Board of Directors, serve as Chairman of the

Board and receive an annual base salary during 2002 of \$433,000, increasing each year by at least 10% of the preceding year's base salary. Dr. Berger is eligible each year to receive a discretionary bonus, determined by the Board of Directors, of up to 50% of his annual base salary. If we fail to renew the employment agreement, we are obligated to pay Dr. Berger, in addition to his compensation for the remainder of the term, a lump sum payment equal to two times Dr. Berger's annual salary for the final year of the term and to provide for the immediate exercisability of all stock options and other equity rights.

Dr. Berger's employment agreement provides that, if the agreement is terminated by either party upon the occurrence of certain events, including (i) our sale or merger (or stockholder approval of a merger agreement) or an acquisition of a substantial equity interest in us by a person or group of persons, (ii) if Dr. Berger is not elected to membership on our Board of Directors, named as Chairman or designated as Chief Executive Officer or ceases to be our highest ranking executive officer or ceases to control personnel decisions with respect to our employees, (iii) if we are in material breach of the terms of his employment agreement, (iv) if we are bankrupt or insolvent or (v) if we terminate Dr. Berger's employment agreement without cause, (1) we will pay Dr. Berger the greater of (x) any remaining salary payable during the term of the agreement plus the maximum possible bonus for each year remaining in the term (taking into account, in both cases, obligated 10% increases in salary) and (y) an amount equal to twice his current annual salary and maximum bonus for the current year of employment (the "Severance Payment") and (2) all of his stock options, stock awards and similar equity rights will immediately vest and become exercisable. We are not obligated to make the Severance Payment if we discharge Dr. Berger for cause. If the vesting of certain benefits and the payment of certain amounts by us to Dr. Berger are treated as payments in the nature of compensation that are contingent on a "change in control" (within the meaning of Section 280G of the Internal Revenue Code of 1986, as amended (the "Code")), the deductibility of such payments could, depending upon the aggregate amount of such payments, be disallowed pursuant to Section 280G of the Code and an excise tax could be imposed on Dr. Berger pursuant to Section 4999 of the Code for which he would, pursuant to the employment agreement, be indemnified by us on a net after-tax basis. The employment agreement contains a non-competition provision that is effective during the term of the agreement and, if Dr. Berger is terminated for cause, for a period of one year following the date of termination.

We also entered into employment agreements with Mr. Berstein, Mr. Casselman and Dr. Iuliucci. The agreements provide for employment through December 31, 2003 for Mr. Berstein and Dr. Iuliucci, and through February 28, 2004 for Mr. Casselman, at annual base salaries during 2002 of \$242,000, \$242,000 and \$227,000, respectively, increasing each year by an amount to be determined by the Board of Directors. In addition, each executive is eligible each year to receive a performance bonus, to be determined by the Board of Directors, of up to 30% of his annual base salary, which may be paid in the form of deferred compensation under the 1997 Executive Compensation Plan, awards of our stock options, or cash. The agreements are renewable for successive one-year terms with the mutual consent of the parties. Effective March 18, 2002, Mr. Steele resigned as Senior Vice President but has been retained under an agreement which provides for his part-time employment through no later than June 28, 2002.

Our agreements with Mr. Berstein, Mr. Casselman and Dr. Iuliucci also provide that (i) upon a change of control, such officers will be entitled to receive, upon termination by the officer within 90 days after the change in control, any remaining salary payable during the term or six months' salary, whichever is less, and all stock options held by such officers will immediately vest and become exercisable; and (ii) upon termination by us, without cause, such officer will be entitled to receive his current salary for the remaining period of the applicable term and all outstanding options that would have vested during such term shall vest immediately.

## Compensation Committee Interlocks and Insider Participation

During the fiscal year ended December 31, 2001, the Compensation Committee was comprised of Messrs. Smith, Snyderman and Troubh. Pursuant to our stock option plans, each member of the Compensation Committee was awarded options to purchase 15,000 shares of our common stock at \$4.09 per share and 25,500 shares of our common stock at \$5.00 per share.

## ITEM 12: SECURITY OWNERSHIP OF CERTAIN BENEFICIAL OWNERS AND MANAGEMENT

The following table sets forth, as of March 20, 2002, certain information with respect to (i) each person (including any "group" as defined in Section 13(d)(3) of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), known to us to own beneficially more than 5% of our Common Stock, (ii) each of our directors, (iii) each executive officer named in the Summary Compensation Table under "Executive Compensation" and (iv) all directors and executive officers as a group. In accordance with the rules promulgated by the SEC, such ownership includes shares currently owned, as well as shares that the named person has the right to acquire within 60 days of March 20, 2002, including, but not limited to, shares that the named person has the right to acquire through the exercise of any option. Except as otherwise indicated, we believe that the stockholders listed in the table have sole voting and investment powers with respect to the Common Stock shown as beneficially owned by them based on information provided to us by these stockholders. Percentage ownership is based on 32,418,371 shares of our common stock outstanding as of March 20, 2002.

### Stock Ownership by Management, Directors and 5% Beneficial Owners

<u>Beneficial Owner</u>	<u>Number and Nature of Shares Beneficially Owned</u>		<u>Percent of Class</u>
Ironwood Capital Management, LLC	2,195,800	(1)	6.8%
Laurie A. Allen, Esq.	135,000	(2)	*
Harvey J. Berger, M.D.	1,787,747	(3)	5.4%
David L. Berstein, Esq.	134,415	(4)	*
Fritz Casselman	52,329	(5)	*
John D. Iuliucci, Ph.D.	251,286	(6)	*
Brian A. Lajoie	2,000	(7)	*
Vaughn D. Bryson	140,500	(8)	*
John M. Deutch, Ph.D.	140,053	(9)	*
Jay R. LaMarche	449,356	(10)	1.4%
Sandford D. Smith	156,705	(11)	*
Ralph Snyderman, M.D.	173,000	(12)	*
Raymond S. Troubh	166,749	(13)	*
All directors and executive officers as a group (12 persons)	3,589,140	(14)	10.5%

\* Indicates less than one percent of the outstanding shares of common stock.

- (1) Such shares are held of record by Ironwood Capital Management, LLC, located at 21 Custom House Street, Suite 240, Boston, Massachusetts 02109. This information is based solely on review of schedule 13G, which was filed with the SEC on February 14, 2002.
- (2) Consists of 135,000 shares issuable upon the exercise of stock options.
- (3) Includes 416,500 shares issuable upon the exercise of stock options. Includes 771,428 shares of common stock held of record by the Berger Family Trust and 8,928 shares of common stock held of record by the Wolk Family Trust. Wendy S. Berger and Harvey J. Berger, as co-trustees of such trusts, have the right to vote and dispose of the shares held by such

trusts; however, in certain circumstances, Wendy S. Berger as co-trustee will have sole voting power with respect to the shares held by each such trust. Includes 120,000 shares held by Edith Berger, Dr. Berger's mother, 40,892 shares held by Wendy S. Berger, Dr. Berger's spouse, and 13,928 shares held by Dr. Berger's children. Dr. Berger's address is c/o ARIAD Pharmaceuticals, Inc., 26 Landsdowne Street, Cambridge, Massachusetts 02139. Dr. Berger disclaims beneficial ownership of any of the 955,176 shares held by the Berger Family Trust, the Wolk Family Trust, Edith Berger, Wendy S. Berger and Dr. Berger's children.

- (4) Includes 131,964 shares issuable upon the exercise of stock options.
- (5) Includes 50,000 shares issuable upon exercise of stock options.
- (6) Includes 240,428 shares issuable upon the exercise of stock options.
- (7) Consists of 2,000 shares issuable upon exercise of stock options.
- (8) Includes 98,000 shares issuable upon the exercise of stock options.
- (9) Includes 105,500 shares issuable upon the exercise of stock options.
- (10) Includes 145,607 shares issuable upon the exercise of stock options and 6,696 shares held by Carol B. LaMarche, Mr. LaMarche's spouse.
- (11) Includes 128,357 shares issuable upon the exercise of stock options.
- (12) Includes 63,000 shares issuable upon the exercise of stock options.
- (13) Includes 110,500 shares issuable upon the exercise of stock options.
- (14) Includes 1,626,856 shares issuable upon the exercise of stock options held by all directors and executive officers as a group.

### **ITEM 13: CERTAIN RELATIONSHIPS AND RELATED TRANSACTIONS**

Our subsidiary, AGTI, holds licenses from Harvard University, Stanford University and other universities relating to our ARGENT cell-signaling regulation technology. Minority stockholders of AGTI, including Harvard University, Stanford University, some of our scientific advisors, and some current and former members of our management, own 20% of the issued and outstanding capital stock of AGTI. We own the remaining 80% of the issued and outstanding capital stock of AGTI. Four members of our management team and/or Board of Directors own or have the right to acquire up to approximately 6.1% of the outstanding capital stock of AGTI. Harvey J. Berger, M.D. owns 3.4%; David L. Berstein, Esq. owns 0.3%; John D. Iuliucci, Ph.D. owns 0.7%; and Jay R. LaMarche owns 1.7%. AGTI has a right of first refusal on the sale to third parties of 73% of the minority stockholders' AGTI shares. AGTI does not have a call option, or a right to require the minority stockholders to sell their shares to us, for any of these shares. As part of the formation of AGTI, we entered into agreements with AGTI to provide for the operations of AGTI.

## PART IV

### ITEM 14: EXHIBITS, FINANCIAL STATEMENT SCHEDULES AND REPORTS ON FORM 8-K

(a)(1) The following Consolidated Financial Statements, Notes thereto and Independent Auditors' Report have been presented in Item 8:

Independent Auditors' Report

Consolidated Balance Sheets

Consolidated Statements of Operations

Consolidated Statements of Stockholders' Equity

Consolidated Statements of Cash Flows

Notes to Consolidated Financial Statements

(a)(2) Financial Statement Schedules:

Schedules have been omitted because of the absence of conditions under which they are required or because the required information is included in the financial statements or notes thereto.

The Exhibits listed in the Exhibit Index are filed herewith in the manner set forth therein.

(b) Reports on Form 8-K

We filed a Current Report on Form 8-K on October 9, 2001 announcing that we will present an update of our product development portfolio and business development initiatives at the UBS Warburg Global Life Sciences Conference on October 11, 2001.

We filed a Current Report on Form 8-K on October 11, 2001 announcing that results of recent studies on our new small-molecule drug candidates to treat bone metastases and solid tumors and the status of their development are being presented at the UBS Warburg Global Life Sciences Conference.

We filed a Current Report on Form 8-K on October 12, 2001 announcing that new data of our lead product candidates to treat bone metastases and osteoporosis are being reported at the annual meeting of The American Society for Bone and Mineral Research.

We filed a Current Report on Form 8-K on December 7, 2001 announcing that results of preclinical studies comparing the safety profile of our regulated erythropoietin (Epo) product candidate with that of an uncontrolled version of Epo therapy in animal models are being presented at the annual meeting of the American Society of Hematology.



## SIGNATURES

Pursuant to the requirements of Section 13 or 15(d) of the Securities Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized, in the City of Cambridge and Commonwealth of Massachusetts on the 22nd of March, 2002.

### ARIAD PHARMACEUTICALS, INC.

By: /s/ Harvey J. Berger, M.D.

Name: Harvey J. Berger, M.D.

Title: Chairman, Chief Executive Officer, and President

Pursuant to the requirements of the Securities Exchange Act of 1934, this report has been signed below by the following persons on behalf of the registrant and in the capacities and on the dates indicated.

Signature	Title	Date
<u>/s/ Harvey J. Berger</u> Harvey J. Berger, M.D.	Chairman of the Board of Directors, Chief Executive Officer, President (Principal Executive Officer)	March 22, 2002
<u>/s/ Sanford D. Smith</u> Sanford D. Smith	Vice Chairman of the Board of Directors	March 22, 2002
<u>/s/ Brian A. Lajoie</u> Brian A. Lajoie	Interim Chief Financial Officer (Principal Financial and Accounting Officer)	March 22, 2002
<u>/s/ Vaughn D. Bryson</u> Vaughn D. Bryson	Director	March 22, 2002
<u>/s/ John M. Deutch</u> John M. Deutch, Ph.D.	Director	March 22, 2002
<u>/s/ Jay R. LaMarche</u> Jay R. LaMarche	Director	March 22, 2002
<u>/s/ Ralph Snyderman</u> Ralph Snyderman, M.D.	Director	March 22, 2002
<u>/s/ Raymond S. Troubh</u> Raymond S. Troubh	Director	March 22, 2002